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TRANSACTIONS

OF THE

AMERICAN OTOLOGICAL SOCIETY,

FIFTY-FIFTH ANNUAL MEETING

HOTEL RALEIGH

WASHINGTON, D. C.

MAY 1, 2 and 3, 1922

VOLUME XVI. PART I.

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The fifty-fifth annual meeting of the American Otological Society was held at the Hotel Raleigh, Washington, D. C., (the Wednesday morning session being held at the building of the District of Columbia Medical Society) May 1st, 2nd and 3rd, 1922.

The following members and guests registered at the meeting:

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DR. G. C. ALBRIGHT
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DR. J. K. M. DICKIE
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Business Meeting.

Tuesday, May 2nd, 9 a. m., The President, Dr. H. S. Birkett, Presiding.

The Minutes of the last meeting were read, and after correction, order approved.

The following nominations for membership were made:

Dr. J. K. M. Dickie, Ottawa. Proposed by Dr. J. G. Wilson; seconded by Dr. J. F. McKernon.

Dr. Lewis Fisher, Philadelphia. Proposed by Dr. T. J. Harris; seconded by Dr. S. MacCuen Smith.

Dr. Perry Goldsmith, Toronto. Proposed by Dr. E. R. Lewis; seconded by Dr. E. B. Dench.

The Secretary presented his report as Treasurer of the Society, for the year just ended.

Upon motion, duly seconded, the report was ordered received and placed on file.

Upon motion, duly seconded, the President was authorized to appoint an auditing committee to audit the Treasurer's books. He appointed Drs. Emerson and Lewis.

Treasurer's Report

Expenditures for 1921-1922.

June 7, 1921	V. B. Alvarez (Expenses at Atlantic City)	$30.50
June 15, 1921	Alfred Moore (Lantern)	15.00
June 16, 1921	Postage	4.00
June 27, 1921	V. B. Alvarez (Reporting Annual Meeting)	95.06
Nov. 5, 1921	Clerical Hire	50.00
Jan. 9, 1922	Postage	6.65
March 2, 1922	Mercury Publishing Co	784.56
April 19, 1922	Postage	2.00
April 19, 1922	Thomas J. Harris (Bank Exchange for the year)	2.42
Total		$1013.94

1922—April 29th

Balance brought forward from June 1, 1921	$ 304.77
Receipts during year June 1, 1921 to April 27, 1922	1479.00
	$1783.77
Expenditures as above	1013.94
Balance	$ 769.83

Dr. Dench, as Chairman of the Committee on the Standardization of Tuning Forks, presented the following report:

The Committee recommends for functional examination:

First: A low fork standardized which, clamped, must give as a lower limit 26 double vibrations per second and varying pitches beyond this to 64 double vibrations unclamped.

For the testing of bone conduction either a C. 256 or C. 572 fork, whichever is the better made, with the addition of a broad base.

The ordinary Galton whistle for the determination of the upper tone limit. This instrument while inaccurate from the point of view of the physicist, is the instrument most available.

The Committee proposes to take steps to have these instruments more accurately made, and this can probably be done.

It is to be understood that this is the minimal equipment and that great value can be obtained from intermediate forks. The cost of the minimal equipment will be about $25.

The Committee suggests that it be continued and at the next meeting a proper blank be presented for recording the results of functional examinations.

EDWARD B. DENCH, M. D.
Chairman.

Upon motion, duly seconded, the report was ordered received and placed on file.

Upon motion, duly seconded and carried, the Committee was ordered to be continued and to report at the next annual meeting.

Discussion:

Dr. E. A. Crockett said he did not want to criticise the deliberations of the Committee, but he did not consider the Galton Whistle much good; nothing short of the Edelman was worth while.

Dr. Dench said the Committee had been up against the fact that it was impossible for everybody to purchase an Edelman at $40., also a set of Kernig rods at $35. That put the equipment out of reach of the younger men.

Dr. McKernon said he had worked on the Galton Whistle a good deal, and he had brought out a purer tone; if one substituted 8 inches of rubber tubing instead of the bulb, it diminished the air puffing.

Dr. Shambaugh said that with a tube and mouth piece one got rid of the puffing sound.

Dr. Randall said he had had many whistles made in Philadelphia and had originated a Galton Whistle in the physical laboratory of the University of Pennsylvania, which closely approached the Galton. The ordinary commercial whistle made was a mongrel.

The Committee on Oto-sclerosis reported, and requested that the Committee be continued.

The Committee on the Deaf Child reported progress. Dr. Richardson, as Chairman, asked that the Committee be continued, to present a report at the next annual meeting.

On motion, duly seconded, and carried, the above committees were ordered to be continued.

Dr. Richardson reported for the Committee on Arrangements. It was moved, seconded and carried, that the Committee of Arrangements be thanked for its services.

Upon recommendation of the Council, it was moved, seconded and carried that an assessment of $15. per member be made for current expenses.

Report of the Necrologist.

The Necrologist, Dr. B. Alex. Randall, reported the death of Dr. John Orne Green. The following notice, prepared by Dr. Henry Lee Morse, was presented:

JOHN ORNE GREEN, (June 7, 1841—Jan. 5, 1922).

The Society has suffered this year the loss of one of its oldest members, our former President, Dr. J. O. Green, who was probably known to all too few of the present membership. One of the founders, he served nine years as Secretary-treasurer, the second of the three who, in that capacity, guided the affairs of the Society through its first thirty years, and in 1881-2-3, was its President. An A. B. of Harvard in '63, he graduated from its Medical School in '66 and spent several years in the study of Aural Surgery in Berlin, Vienna and Wuertzburg. Settling in Boston and serving on the Staff of the City, the Masachusetts General and the Eye and Ear Hospitals, he early gave instruction in our specialty and became Clinical Professor of Otology in Harvard in 1888-94. He contributed in '98 the chapter "Operation on the Ear" to the American Textbook of the Diseases of the Eye, Ear, Nose and Throat, and a few other masterly papers to our literature; but retired in 1898 and was all too rarely present at our meetings. Yet a man so sane and competent lives long and valuably in the ideals and work of his pupils and associates; and Orne Green must be counted one of those modest worthies who lives on in the achievements of many followers, who may but partially have realized the formative influence of his teaching. I met him but four or five times, yet would claim to be one of his disciples.

One who knew Dr. Green well, and worked with him for years, said of him: "Professionally he was a careful, painstaking, and skilful surgeon, who gave the very best thought and care to every case that came before him; and as a man, he was a modest, kindly and delightful gentleman, with whom it was a great pleasure to be associated."

Upon motion duly seconded and carried, it was moved that as Dr. Green had no family, a copy of the report be forwarded to the Trustees of the Eye and Ear Infirmary at which institution Dr. Green had been an active worker.

A recommendation from the Council was then made in regard to the establishment of a special journal dealing with otolaryngology. The trustees of the A. M. A. were considering favorably the editing of such a publication, if a request for same were received from the three societies and their endorsement were obtained. The Council favored such an endorsement of the project.

Dr. Shambaugh said this matter had come up before, and the objections which held in the past had disappeared. These related to the financial question. Now it was felt that the editors did not want to publish the Transactions of the Society in the general columns of the Journal of the A. M. A. and would rather have a special journal to deal with it. The establishment of a high class journal would stimulate the ideals of the younger men. Finally the A. M. A. would assume financial and editorial responsibility.

Dr. Dench asked how this would affect special journals now in existence. The Annals of Otology and the Laryngoscope had done excellent work. Was there enough good material for three special journals.

Dr. Shambaugh said that the field for a new journal would quickly become clear, and that the old journals to which they owed a debt of gratitude would not be discouraged or detracted from.

Dr. Dench said that it was frankly a matter of having a journal of a higher type.

Dr. Shambaugh said that the new A. M. A. building was now complete and that there was room for conducting new journals, which made it a favorable time for societies who desired a special journal to bring forward their request.

Dr. Randall said that the Otological Society was the senate of otological interest in the country, and should stand for the highest type of publication. He favored offering an invitation to the A. M. A. to publish such a journal.

The matter of further unifying the subjects of otology and laryngology was brought forward by Dr. Birkett, who suggested that a sub-committee be appointed by the Otological Society to confer with a similar committee of the Laryngological Society, to report at the next Annual Meeting.

It was moved, seconded and carried that the Chair be authorized to appoint such a committee.

The Chair announced that Drs. McKernon, Jack and MacCuen Smith, were appointed to serve on this committee.

The Council, acting as a Nominating Committee made the following report:

President—Dr. G. E. Shambaugh, Chicago.

Vice-President—Dr. John B. Rae, New York.

Secretary-Treasurer—Dr. Thomas J. Harris, New York.

Additional Member of Council—Dr. L. W. Dean, Iowa City.

It was moved, seconded and carried that the report be received and placed on file.

The following men were recommended by the Council for active membership in the Society:

Dr. Wesley C. Bowers, New York.

Dr. H. P. Cahill, Boston.

Dr. Clifton L. Miller, Richmond, Va.

It was moved, seconded and carried that the Secretary be instructed to cast one ballot for the candidates named. This being done they were thereupon declared duly elected.

The Secretary called attention to the coming International Otological Congress in Paris, and said that any members preparing to go, would, upon application be given certificates as delegates.

The resignation of Dr. Crouch of Baltimore was presented.

It was moved, seconded and carried that the resignation be accepted.

Dr. Emerson for the Auditing Committee reported that the books of the Treasurer had been examined and found correct.

It was moved, seconded and carried that the report of the Council as Nominating Committee be adopted and the Secretary be instructed to cast one ballot for the officers named.

The Secretary reported that this was done, thereupon the Chair declared them duly elected.

It was moved, seconded and carried that a letter of thanks be sent by the Secretary to the District of Columbia Medical Society for their courtesy in lending their building for the meeting.

The Chair appointed Dr. Crockett a Committee to escort the incoming President to the platform.

Dr. Shambaugh, in a brief speech, expressed his appreciation of the honor done by the Society in electing him President.

The meeting was then declared adjourned.

REPORT OF FIVE CASES OF MASTOIDITIS WITH ATYPICAL SYMPTOMS.

By WENDELL C. PHILLIPS, M. D. and ISIDORE FRIESNER, M. D.,
New York City.

The purpose of this paper is to call attention specifically to those cases of purulent mastoiditis which do not come under the usual classification and do not present its so-called typical symptoms. By this we refer to the drooping of the posterosuperior canal wall, tenderness on pressure over the mastoid cortex, deepseated and continuous pain, fever, continuous discharge from the external auditory canal, and postauricular swelling. It seems timely, therefore, again to call attention to the fact that there are instances of osteitis of the mastoid process requiring operative interference which are secondary to tympanic infection but in which the tympanic portion of the disease, so far as signs are concerned, plays a comparatively insignificant rôle.

In the cases about to be described, the history shows no discharge at any time, or at least no recent discharge, from the middle ear. Definition of diseases in textbook form must necessarily be more or less dogmatic in character; hence our clear-cut textbook description of the indications for the mastoid operation. But as our studies have been more thorough and more complete, particularly by men of wide experience in special hospitals, we have been forced to the recognition of a rather large proportion of atypical cases, and believe that these cases are far more common than we might otherwise suppose. In other words, there are many operative cases in which the majority of the so-called typical symptoms are absent. Many times patients present themselves with characteristics of the middle ear and external auditory canal indicating the presence of an operative mastoiditis who perhaps at some previous time may have had some of the so-called classical signs and symptoms of mastoid disease. When, however, they come

under observation, they have, with the exception of a middle ear that gives no evidences of resolution, no other cardinal signs or symptoms of mastoid involvement.

The peculiarities or the peculiar appearance of these middle ears has recently been described by Dr. Dench. In children, particularly when the history of headache or pain is not obtainable, it has frequently been our experience to see a subperiosteal abscess as the first evidence of mastoid disease or of middle ear infection. Occasionally such a subperiosteal abscess will not be tender. It is true, of course, that this condition is invariably secondary to an infection of the middle ear, and in no case in which we have observed this state of affairs have we been able to conclude that we were dealing with perfectly a normal tympanum. Such instances, however, have been reported, but we are doubtful whether the observers had taken into account minor changes in the drum, slight fulness or thickening, both of which are evidences of recent tympanic infection. Too much stress has been put upon the classical signs and symptoms of mastoiditis, and too little is known by our graduates in medicine concerning severe mastoid infections in which most of the so-called typical symptoms are absent. It is a curious fact that patients are frequently admitted to the services in general hospitals with unilateral headache and tenderness over the mastoid without any discharge from the ear, and the condition present is not suspected because of the absence of a discharge from the ear. The following cases represent the types which we have in mind:

Report of Cases.

Case 1.—E. L., a man; more than two months before admission into the hospital had a cold in his head, followed by pain and tinnitus and partial deafness in the right ear. This lasted two weeks and then the pain subsided, but the deafness persisted. There was no discharge. A week or two before admission, the pain in the right ear again became severe and extended over the right side of the head. There

was some fever, but still no discharge. A few days before admission, the patient had a distinct chill. The left mastoid had been operated on a year previous. The right canal was distinctly narrowed. The drum was thick, but not bulging. There was no perforation and no discharge. No landmarks could be made out. The mastoid was tender far back over the emissary. At operation an enormous perisinous and epidural abscess was found.

Case 2.—E. H., a man, in September had influenza. During this attack he had some pain in the right ear, lasting a few days. He was confined to bed one week. When he was up and about again he complained of tinnitus and some stuffiness in the right ear. At no time was there any discharge. Early in October he consulted an able otologist, and during the months of October, November and December was under his care. The treatment consisted of inflation, the usual care of a tubotympanic catarrh. All this time, however, the patient complained of unilateral headache. December 31, he consulted one of us. At that time there was a narrowing of the canal toward the tympanum. The drum was thick and lusterless, but not bulging. The short process could be seen as well as a little of the hammer handle. There was no tenderness over the mastoid proper, but somewhat farther back there was distinct tenderness over the region of the sinus. The diagnosis of perisinous abscess was made, and an operation was advised. A large perisinous and epidural abscess was found. No myringotomy was performed. Healing was uneventful. The patient was discharged at the end of nine weeks, with hearing of twenty-six double vibrations, whisper at 25 feet and positive Rinné test.

Case 3.—D. B., a man, had been treated for nose trouble, probably sinusitis, but without apparent ear complications, nine months before. Five weeks before he had a severe attack of pain in the left ear and the left temporal region. External swelling soon became marked, and he sought relief in the ear service of the Manhattan Eye, Ear and Throat Hospital. The external swelling was so extensive,

partially closing the external auditory canal, that the majority of the staff considered it to be furunculosis. He then consulted an otologist at his office, who opened the ear drum, but no discharge followed. At no time during the subsequent treatment was there any discharge from the external auditory canal. The external swelling extended anteriorly until it involved both left eyelids; when we saw him, both eyelids were edematous. At this time a roentgenogram disclosed extensive involvement of a large pneumatic mastoid. Arrangements were made for operation, but the man disappeared for about a week ,reporting that the external swelling had subsided. It returned, however, and became so extensive that the left eye was entirely closed and the eyelids of the right eye were considerably swollen and edematous. Mastoid operation was then performed, and a large cortical perforation was found at the root of the zygoma. An enormous abscess involved the soft tissue of the temporal and parietal region of the scalp. He also had a very large perisinous abscess, and the dura of the middle cerebral fossa was denuded over a wide area. He made a rapid and uneventful recovery, with good hearing.

Case 4.—Dr. Hays, of the Section on Otology, New York Academy of Medicine, April 14, 1922, reported the history of a child, aged 4 years, who had temperature of about 102 F. for three days, accompanied by grippal symptoms. Then there was a mild attack of scarlet fever with a temperature of about 103° for a few days longer, when the temperature became normal. Three weeks later the attending physician, a pediatrician, found a large cervical gland on the left side of the neck, immediately below the mastoid bone, and a temperature of 103°. There was no complaint of pain, but examination revealed the external auditory canal so swollen that it was impossible to obtain a view of the drum. Mastoid tenderness was present, but the child did not appear to be ill and did not complain of pain. Mastoiditis being suspected, a roentgenogram was made which showed evidences of breaking down the mas-

toid cells. Operation disclosed a large quantity of pus in the mastoid, and a perisinous and epidural abscess. Immediately after operation the temperature became normal, and convalescence was rapid with good hearing. The ear drum was not punctured, and there never has been any discharge from the external auditory canal.

Case 5.—A. R., a woman, early in December had had pain and tinnitus in the right ear. At first the pain was severe and fairly constant; but after the administration of some drugs by the local physician the severe pain ceased. The tinnitus, however, continued, and at intervals during the day there were shooting pains over the right side of the head. There was never at any time discharge from that ear. For two days before coming to the office she noticed a small swelling far back behind the mastoid region. The left ear was normal. The right tympanum was very much thickened so that no landmarks were visible. Hearing in the left ear was normal. The Weber test was referred to the right ear, the low tone limit raised and the hearing for whisper considerably diminished. January 19, simple mastoid operation was performed. The mastoid was extensively diseased. A firm, beet-like, organized granuloma shut off the antrum out of the aditus. Corresponding to the swelling behind the mastoid there was a perisinous abscess in which the sinus lay exposed, covered, however, with granulation tissue for about 1 inch behind the knee, and 1¼ inches in front of the knee. The wound healed rapidly. She heard the whisper at 20 feet, and twenty-six double vibrations; the Rinné test was positive; the Weber test was referred to the right. The postauricular wound was healed in a firm scar. The patient was seen again one year later, and during the interval had no trouble with the right ear.

Five similar cases were seen within six weeks of the one occuring in 1919. Two of these cases occurred in private practice. Three of the patients were operated on in Dr. Phillips' service at the Manhattan Eye, Ear and Throat Hospital.

Conclusions

We feel justified in bringing this to the attention particularly of the teachers of otology. The observation of so many cases of this nature in so short a time is, to our minds, an evidence of the frequency with which they occur. Furthermore, the fact that in virtually every instance no diagnosis was made or even suspected until the patients came into the hands of the otologists seems to us an evidence of the fact that both textbooks and teachers are wanting in their failure adequately to describe this condition to students.

Discussion.

Dr. E. A. Crockett, Boston: I think that these cases that Dr. Friesner describes are extremely important to the otologist.. One explanation of these atypical cases exists in the infantile type of mastoid with a small cortex and single antral cell. There is a thick outer cortical wall. These cases cannot occur in the pneumatic type of mastoid. Where the X-ray shows you an infantile type of mastoid the obscure symptoms may be referrable to this cause. A case I had last year is an illustration of the extreme condition this variety of case may come to. The patient was a doctor's daughter, a girl of 12. The father took care of the case. She was suffering from earache which lasted for a few hours and then subsided. Two weeks later she had malaise, headache, temperature, vomiting and had to be taken out of school. She was sent to the country to recuperate. She was then seen by a well known pediatrician, who said the ear drum was normal, but noted that the child dragged the leg on the same side as the affected ear and asked of there was any connection. He sent the child in for further examination and she presented double optic neuritis and typical cerebellar ataxia with depressed pulse rate. A deep cerebellar abscess was found with streptococcus mucosus in pure culture, but no capsule. The child made an uneventful recovery.

The pediatrician was willing to call for help in this obscure case. Some of these cases escape observation altogether and die of acute cerebro-spinal meningitis. This class of cases is the prime reason for early and repeated X-ray examination, which will show you that there is something wrong. Medical examination will show nothing. X-raying children is difficult because the child won't keep still and that makes the plate cloudy, but still the X-ray will reveal points that would escape observation. Another point is the avoidance of paracentesis in mastoid operation when you have a perfectly free drainage by the attic and antrum cell. You have a better drainage this way than by the tympanum. The cases do just as well as with a routine paracentesis in the course of the operation.

Dr. J. G. Dwyer, New York: What organisms were found in that series of cases?

Dr. I. Friesner: I don't remember all of them. They were not all mucosus; one was a pneumococcus and two were hemolytic streptococcus.

Dr. Thomas J. Harris, New York: Drs. Phillipps and Friesner have touched a chord which will appeal to every man doing otological surgery. Every one of us has encountered just such cases. The subject is not a new one. It has been written about from time to time in the last 14 or 15 years. To make the conclusions of Drs. Crocket' Phillipps and Friesner of real value, we should like to hear some deductions. We all recognize that there are atypical cases; hundreds are reported, but will they tell us what we can deduce from this study. Dr. Crockett made a valuable deduction in regard to the X-ray pictures, but we wish he would tell what he thinks about disregarding classical symptoms; and in the absence of these,— the drooping of the canal wall and tenderness over the mastoid, what weight does he attach to the type of bacterial organisms? I think the subject is old enough and the number of cases large enough to draw definite conclusions as to distinct

symptoms which point to mastoid involvement and demand operation; or have we reached the point where we have decided that they are not of leading value and can be disregarded? Some men feel that we have put too great weight on these signs; one worker states that drooping of the canal wall is exceptional. It does not do for us to wait until the patient's life is in danger.

Dr. F. T. Hopkins, New York: Was there any noticeable diminution of hearing in these cases?

Dr. Norval H. Pierce, Chicago: The answer to this inquiry we might say is the work of Wittmaack. The normal pneumatization of the temporal bone means that the entire mastoid is occupied by air cells which have very thin walls, which are covered by muco-periosteum, which shows under the microscope an endothelioid epithelium, very thin, and arranged in one or two layers. The sub-epithelial tissue cannot be separated into layers, but constitutes one very thin homogeneous layer. When this type of mastoid becomes inflamed it is usually by the streptococcus mucosus capsulatus, there is swelling of the delicate muco-periosteum, and at a given time—about three weeks—we get a decalcification of the bony walls of the pneumatic spaces and an abscess formation, one cell breaking into another, and in this way extension and inflammation in the direction of the sinus, or it may be upwards and backwards to the meninges. In other words, the extension of inflammation in normal pneumaticized mastoids is by continuity and contiguity of this softening process. This is the classical mastoid picture, beginning with pain and slight infiltration, in any case of severity, over the mastoid fossa, which usually disappears in a day or so, and if the decalcification occurs this pain on pressure recurs at the end of two or three weeks. We usually have periosteal infiltration as an accompanying manifestation of mastoid suppuration in this type of mastoid. On the other hand, if pneumatization has been arrested to any

considerable extent (and this arrest occurs with far greater frequency than we have hitherto believed) then we are apt to have quite a different pathological process. Instead of a thin muco-periosteum we have a thick muco-periosteum, and instead of the endothelioid epithelium, we have a thick, more cuboid epithelium or ciliated epithelium, and a thick sub-epithelial tissue with more or less of the old myxomatous tissue which formerly filled the cavum and antrum in fetal life. This thick tissue serves to protect the bone against inflammatory softening, while it is more liable to inflammation than the normal muco-periosteum. It is richer in blood supply and the thick bone underneath is resistent to the softening, decalcifying process. It has low grade vitality compared to the normal tissue, and is subject to inflammation tending to chronicity. These cases produce the chronic running ear with and without cholesteatoma.

How do complications occur in these cases when arrest of pneumanitization is marked? They do not occur by continuity of the softening process as in the normal mastoid. They are blood-borne inflammations or osteophlebitic, and it is a characteristic of these arrested pneumanitization cases that there are abnormal vascular communications between the thick walled pneumatic spaces, the meninges, the lateral sinus and the jugular bulb.

We must study these cases more intensively. In my experience they constitute the atypical mastoid cases, and the lesson we can carry away is what Dr. Crockett emphasized; that is, to pay more attention to the x-ray findings. It is my opinion that in every case of suppurative otitis media of any severity, the x-ray should be employed at the very outset as a routine matter, before there are symptoms referable to the mastoid. In that way we can tell whether we have arrested pneumatization or if we have the normally pneumatized mastoid, and we can watch the progress of the disease and in many cases foretell its progress.

Dr. Emil Amberg, Detroit: I would like to ask what the blood count revealed in the case reported.

Dr. Edward B. Dench, New York: The whole question of atypical mastoid cases is a very interesting one. Very soon the atypical cases will be the typical ones, and vice versa. I remember a case of a young boy who four weeks before he was admitted to my ward at St. Luke's, had earache two days. His temperature was 106° on admission. He was perfectly happy, had no pain and complained of nothing except slight tenderness at the angle of the jaw. There was no mastoid tenderness on either side and the right drum membrane appeared normal. On the left side there was a slight sinking of the upper wall of the canal, it was very slight and within normal limits. The X-ray showed slight cloudiness on both sides. The white blood count was 15,000, polys 73 per cent. The temperature then fell to 101° and I was congratulating myself that I had not done an unnecessary operation, when the temperature shot up to 106° again. I decided to put a ligature above the left internal jugular, and I tied off the facial vein as well. I then rapidly opened the mastoid, and in spite of the X-ray which I had taken to the best man in New York for interpretation to confirm my opinion, I found the mastoid completely broken down and there was a perisinus abscess which did not show in the plate. Having tied off the jugular I curetted out the sinus, and I opened the other mastoid and found that also broken down. The boy made a complete and perfect recovery, the temperature fell immediately and he went out of the hospital perfectly well. We do not incise the drum membrane in late cases with cloudy X-ray. I am glad Dr. Crockett brought out that point. It should be for exploratory purposes rather than routine practice. I think Dr. Pierce's explanation is an excellent one in so far as it is an embolic process. That is the only explanation for extensive destruction in a short time. The other cases are from continuity of structure. The X-ray is very definite in the cases, but when infection is through the blood stream with change from breaking down, it is very deceptive.

In regard to mastoid cases, we open them sometimes and find the mastoid filled with granulation tissue and at a certain point the tissue acts as a support to the walls of the cells. The mastoid cells are perfectly definitely marked, and the outline of the cells is as distinct as if there were no inflammation there. We don't know whether the case is going on to resolution or to breaking down. I am heartily in accord with the routine practice of the X-ray. Four weeks ago we had a case where the mastoid showed no change, but later there was clear evidence of breaking down. This illustrates the necessity in all cases of acute otitis, of taking repeated X-ray pictures. It is a wise thing because you can compare pictures at the time of the first acute attack with those of the second attack.

Dr. Robert Lewis, New York: I would like to make a different observation. My experience does not coincide with Dr. Crockett's. I believe these atypical cases occur more often in the large pneumatic type of the mastoid process.

Dr. Wendell C. Philipps: I operated on one of these cases and it was the largest penumatic mastoid I ever operated on. In regard to Dr. Harris' observation, I don't know whether we can draw any deductions or not. These cases are frequently treated in the general hospitals and they do not primarily come to the otologists. In regard to the teaching standpoint, we ought to teach that these cases do occur, and that one should always be suspicious of unilateral headache.

Dr. S. MacCuen Smith, Philadelphia: The question of early myringotomy is perhaps the most important part of the treatment of acute tympanic disease. When such disease complicates the exanthemata or other virulent infection, myringotomy should be performed early, even in the absence of definite bulging, from the fact that the membrana tympani is much more oblique in children than

in adults, and during an attack of otitis media it does not necessarily bulge nor is it constantly red. Having undergone certain changes, it takes on a grayish appearance, with obliteration of the landmarks. Under such conditions prompt myringotomy is urgently indicated. Dr. Harris brought up the question of sagging. We appreciate the value of sagging when present. It is an unfailing sign for performing a mastoid operation. Cases do not get well without operation when drooping is present, but it only occurs when the antrum is especially involved, as manifested by necrotic changes in the posterior osseous canal. You may have cases which require operation just as urgently, when the lower cells are involved, without sagging. In acute suppurative otitis media the mastoid is always involved, but not in the sense of a true mastoiditis. Recovery takes place in the majority of cases owing to the fact that the aditus ad antrum remains patulous. I pointed out the value of X-ray examinations to endorse this principle. It will tell us what is going on and enable us to appreciate the progress of the case, and; furthermore, always shows the location of the sinus, which, from an operative viewpoint, is very important.

Dr. George E. Shambaugh, Chicago: I would like to ask Dr. Dench a question which may be very simple for him but which has puzzled me a great deal. It is this—where he has to do with a case of bilateral otitis media and the symptoms point toward sinus thrombosis what would be his procedure? Would he, in this case, ligate the internal jugular before exposing the sinus? Suppose this is done on one side and the exposure discloses no thrombus, would he proceed in a similar manner with the opposite side? Is there harm in ligating the internal jugular on both sides?

Dr. Phillipps: I would like Dr. Friesner to close the discussion.

Dr. I. Friesner (closing): You cannot ligate both jugulars. Dr. Amberg asked about blood counts,—I saw the cases at the same time a few hours before operation and we did not make blood counts. I think Dr. Harris will agree that the discussion has brought out a method of handling these cases, which is what we expected. The point is this, these cases have been reported time and again. It is an old story, yet seeing so many of them going unrecognized, sometimes two or three months after otitis, is some reason for bringing this matter to the attention of men teaching otology, so that they may point it out to students. In regard to the discussion on anatomy by Drs. Pierce and Crockett, on the so-called infantile type of mastoid being most frequently associated with intracranial complications, the work of Wittmaack is beginning to make the reason clear. We have known it clinically for a long time. However, the cases reported were not anatomically of the type Dr. Crockett described. There are three types: first, the large pneumatic type with or without open antrum, in which the antrum is filled with firm organized granuloma; second, the large pneumatic mastoid in which infection takes place by continuity along the cells behind the posterior canal wall. It is plain that extensive destruction can go in the mastoid remote from the antrum since the latter is shut out of the remainder of the mastoid and the antrum and middle ear may clear up; third, the infantile type with small antrum, more or less shut out from the mastoid disease.

Dr. E. A. Crockett: Did you ever see an infection caused by streptococcus hemolyticus

Dr. I. Friesner: Yes, in several instances.

Dr. B. Alexander Randall, Philadelphia: Does the antrum grow after infancy? Are the cases pathological or developmental?

Dr. I. Friesner: I do not know that anyone has made a sufficient study of the pneumatic mastoid and its changes secondary to repeated infection to answer that. I believe it is developmental, not pathological, in the sense that development is arrested, perhaps secondary to some pathological processs in early childhood, involving both mastoid and antrum. I would like Dr. Pierce to give his interpretation of that.

SOME OBSERVATIONS ON THE DIAGNOSIS AND TREATMENT OF AURAL COMPLICATIONS. A PATHOLOGICAL AND CLINICAL STUDY OF OVER 1000 CASES.

By J. G. DWYER, M. D., New York City.

SCHEMA

I. Sinus thrombosis
    A. Diagnosis
        (a) Experimental data
        (b) Pathological
            1. Blood-cultures
            2. Blood counts
        (c) Clinical
    B. Operative treatment
II. Meningitis
    A. Examination of spinal fluid
        (a) Physical
        (b) Chemical
        (c) Cytological
        (d) Bacteriological
III. Brain abscess

The following observations are based upon the experience of the writer during the last twelve years at the Manhattan Eye, Ear and Throat Hospital. During this time, careful clinical and pathological histories of such cases were kept, as during seven years of this time he had charge of the Pathological Department, and the whole intention in writing a report of this kind is to present the whole subject from the purely practical standpoint, endeavoring to clarify some ordinarily obscure conceptions and to be a small aid in the diagnosis and treatment of such cases. The whole is a statement of the practical deductions to be drawn from an intimate study of hundreds of cases, both clinically and pathologically, in addition to data obtained at autopsy and by experimental work.

The majority of the cases were seen by the writer in his position as pathologist and in as many instances as possible, every necessary pathological test was carried out as a routine measure, thus giving us a scientific study in correlation with the clinical study.

The report will deal with the following complications:

(1) Sinus thrombosis

(2) Brain abscess

(3) Meningitis

I.

Sinus-thrombosis

The diagnosis and treatment of sinus-thrombosis are ever present questions with us and the subject will be treated under the following heads, in order to visualize at a glance, the important points.

(a) The diagnosis, (b) the operative treatment, (c) the post-mortem treatment.

A. *Diagnosis:*

The great importance of a proper diagnosis and especially of an early diagnosis, is recognized by all and there is no aural condition that gives us such a varying clinical picture as this condition. It must also be borne in mind that there is a large percentage of cases of sinus-thrombosis that are unrecognized as such and which clear up spontaneously or without operation. This has been proven time and again at autopsy, where death had taken place years afterwards from other causes and only incidentally was a thrombosed or fibrous vein or sinus found. Also, bearing out this contention, we have all seen clinically cases that we are morally sure are thrombosis cases and still the patient recovers without operation. In contrast to these fortunate recoveries we see cases in which the condition had advanced to such a state that recovery is impossible and yet we feel that if the diagnosis had been made early, we could have changed the issue.

Let us then consider the diagnosis under the following sub-divisions: (1) pathological, (2) clinical.

Experimental Work:

Before considering these, we wish to analyze some experimental results obtained in animals and by analogy try to gain an insight into what occurs during the varying stages of sinus-thrombosis in human beings. A series of experiments were carried out on rabbits, producing as far as possible, the identical course of events in the thrombosis of the jugular vein as occurs in lateral sinus thrombosis. By exposing the jugular vein and ligating in two places separated an inch or so, we produced a thrombus in the vein. The ligatures were then removed and the clot or vein walls inoculated with streptococci or staphylococci of known pathogenicity for rabbits. The clinical course was then followed and the changes noted. From these experiments and from clinical and operative experience in human beings, we can visualize for ourselves the pathological course, and taking all these findings in conjunction with each other, we can with fair accuracy outline the probable course of events as follows: there is first set up a phlebitis of the vein wall, which in time is followed by coagulation of the blood and the production of a thrombus. This thrombus may completely shut off the lumen of the vessel or only partially, in the latter case forming the so-called parietal clot. These clots may be sterile in character, although the cause is usually septic in character. The process may end at this point, even if pathogenic bacteria are present, as the normal bactericidal power of the blood may be sufficient to kill off the few bacteria that gain entrance. The clot itself may then be absorbed with consequent reopening of the blood stream, or it may become organized and thus permanently occlude the vessel. In either case, cure results spontaneously. Incidentally, this is what probably has taken place in those cases in which we find the sinus thrombosed, covered with granulations and still absolutely localized, as far as the general system is

concerned and of course here the proper technique is to leave the sinus alone, as nature has already set up better protection than we can give.

If this fortunate outcome does not ensue, the next step is that through the enzyme activities of bacteria, we have a further change in the clot, the latter undergoing coagulative necrosis, which is rapidly followed by liquefactive necrosis and then the flooding of the system at large by bacteria and detritus, and unless the process is checked, death ensues from toxemia, septicemia or pyemia.

Bearing the above observations in mind, let us now consider the pathological diagnosis of sinus-thrombosis. The two important points in this diagnosis are: (1) blood-cultures, (2) blood-counts.

(1) The question of blood-cultures is a very important one and one that should be thoroughly understood so that a proper interpretation can be placed upon it. In the first place, it cannot be too forcibly stated that the average clinical aurist is not in a position to understand blood-cultures unless he takes pains to study up the why and wherefore. By this we mean that again we have seen irreparable delay in operating upon sinus-thrombosis, just because the blood-cultures were negative, the proper interpretation not being placed upon the results. The point to be emphasized here is that negative blood-cultures have no practical meaning on acount of the well recognized limits of blood-culture work and that in the face of a well marked clinical sinus-thrombosis, there should be no delay in the operative procedures, simply because repeated blood-cultures are negative. A lengthy article could be written, giving the reasons why we get negative blood-cultures, where we should expect positive ones, and these reasons are given in any good textbook of bacteriology. Suffice it to say that it is only in a relatively small percentage of the cases that the blood-cultures are positive. The time of taking the blood, the amount of blood, the number of blood-cultures, the media, etc., are all factors and are all well recognized by clinical pathologists. Again, a point

to be emphasized is the fact that blood-cultures may only be positive at certain stages of the disease, or at certain times of the day. The culture is more likely to be positive while the temperature is high than when it is low. The pathological course of a clot as outlined above, will explain positive and negative blood-cultures. Time and again the writer has seen valuable time lost, in the face of a clinical sinus-thrombosis, while the operator waited for a positive report. If operators will fix this one point in their minds, many lives will be saved.

(2) *Blood-counts.* This is very important as a confirmatory point in the diagnosis. Taking an average of hundreds of blood-counts in sinus-thrombosis, the leucocyte count shows an average of 14,500 to 15,000 with a polynuclear average of 78 to 80 per cent. These figures are not of course absolute, but the point to be emphasized is that in the ordinary case, there is a moderate leucocytosis with a relative increase in the polynuclear count. This is a distinct aid, as in ordinary mastoiditis the count is lower, in meningitis and generalized encephalitis is much higher and in localizied brain abscess is practically normal. Again, very often an increase in the percentage of large mononuclear leucocytes and the transitionals is also noted. This latter observation is especially true in the later cases where the thrombosis has lasted for some time.

The foregoing observations on leucocytosis must be based on several blood-counts in each case, and preferrably on repeated daily blood-counts, done as far as possible under the same conditions each day and by the same man. The importance of this cannot be over-emphasized if much confidence is to be placed in the work.

B. *Clinical Diagnosis:*

With the ordinary clinical diagnosis, the clinician is more conversant. The typical chill or repeated chills with the typical septic temperature are looked for and recognized by all, but it is the atypical type, with the high continuous temperature for the first few days and a very slight or no

chill that presents the difficulities. In some of these cases, which, as far as the condition of the patient is concerned, resemble sometimes early meningitis and in others erysipelas, the possibility of sinus-thrombosis must be kept before the mind. Then again we must remember that there are cases that run a practically aseptic course that are liable to throw us off the right track.

II.

The Operative Treatment

The diagnosis having been made, the next question is that of operative procedure, that is, the sequence of events in the operation. Without going into the question too fully the rationale of events that present themselves to our minds is as follows: The cases are to be divided into two classes, (a) with positive blood-cultures, (b) with negative blood-cultures. Each of these classes can be sub-divided into two, those in which the mastoid operation has not been done and those in which it has been done. The procedure in each of these would be in our judgment, as follows, in order to give the patient the best chance.

(1) Suspected mastoiditis and sinus-thrombosis, with positive blood-culture. Perform the mastoid operation, expose the sinus and if found diseased, ligate the jugular vein and then clean out the sinus.

(2) Suspected sinus-thrombosis, with positive blood-culture, in a case already operated on for mastoid, tie the jugular vein immediately and then open and clean out sinus.

(3) Suspected mastoiditis and sinus-thrombosis, with negative blood-culture. Perform the mastoid operation and expose sinus. If found diseased, then tie the jugular as before. If not found diseased, leave alone for observation.

(4) Suspected sinus-thrombosis, with negative blood-cultures in a case already operated on for mastoiditis. Expose the sinus; if found diseased, tie the jugular and then complete the work on the sinus. If not sure of disease, expose sinus but be ready to immediately tie the jugular.

The above is the procedure adopted by the writer for the last few years and he feels it gives the patient the best chance, as it minimizes the danger of flooding the system with broken down clot.

The biggest question in otology is that of sinus-thrombosis in double mastoiditis. All of these cases are difficult but some of the practical points to be kept in mind in deciding on which sinus to tackle are as follows:

(1) The condition of the sinuses at the time of the mastoid operations, that is, whether one is abnormal in any way.

(2) The presence of enlarged glands on one side of the neck.

(3) The palpation of a thrombosed jugular vein.

(4) The feeling on one side of the neck compared with the other. All of these points have been of assistance.

The question as to ligation of the jugular versus excision, is one that has excited much controversy during the last few years. The writer has practiced low ligation, just above the clavicle and sees no necessity or justification for the excision above the facial. Whether we excise above the facial or not, we cannot remove it to the diseased vein and no matter how complete our operation is, we leave some diseased wall which nature usually takes care of. The necessity of removing so much vein has never appealed to the writer and the operation is much simpler and can be performed very quickly.

Meningitis

The subject of meningitis is an all important one to the aurist and the following observations are based upon an experience of over two hundred cases of otitic meningitis with a study of the spinal fluid from over four hundred cases. The writer has for over twelve years been interested in the subject, especially the pathological diagnosis and treatment and hopes he will be pardoned if he goes at

length into some phases of the variations in spinal fluid. The examination of spinal fluid falls under the following heads:

(a) Physical, (b) chemical, (c) cytological, (d) bacteriological.

The physical examination only reveals the fact as to whether the fluid is under pressure or not, and also as to whether it is clear or not. From an analysis of the pressure, the writer has never been able to make a diagnosis of purulent meningitis as compared with other lesions. The pressure in normal individuals varies so much that it is impossible to draw conclusions. The clarity or cleanness is examined under another heading.

(b) *Chemical Examination.* Some years ago an effort was made by one of the members of our Society to decide the presence or absence of early meningitis from the chemical examination of spinal fluid. The main points stressed were the presence or absence of a sugar reducing substance, of choline, ablumen, lactic acid, etc. Suffice it to say that the writer cannot bear out the claims, as the presence or absence of the above substances are too variable to be of much assistance. Certainly they are not reliable enough to justify us in any operative procedure based upon them for absolute diagnosis.

(c) *Cytological.* This has to do with the number and quality of the cells present in spinal fluid. Of late years, many aurists have stressed the number of cells present at the various examinations. The experience of the writer would go to show that not much confidence can be placed in the variations of the cell members as far as prognosis is concerned. Naturally, when a case is recovering the cells will diminish in number, but in a case advancing to a fatal outcome, the varations have been without diagnostic or prognostic significance.

(d) *Bacteriological.* Purulent meningitis, in the series reported by the writer has been caused by the following organisms.

(1)	Streptococcus haemolyticus	89 per cent.
(2)	Pneumococcus (all types)	10 per cent.
(3)	Staphylococcus	1 per cent.

A plea is made here for the sake of exactitude and for the sake of the literature that more care be taken in the diagnosis and nomenclature of meningitis. The writer thinks that no diagnosis of purulent meningitis should be made unless the organism is found in the spinal fluid and cultivated therefrom. If anybody looks up the literature, he will have to throw out case after case that has been described as meningitis, because a close analysis of the case would show that it was not a true case. Much of the fault here lies with the clinicians and much with the bacteriologists, but there is no reason in this age for such a condition of affairs. Thus, several recent reports showed an organism in the spinal fluid that could be stained in direct smear, but could not be cultivated. This is due to the bacteriologist not using the proper technique in recovering organisms from spinal fluid.

Several papers appearing recently have given a differential diagnosis between meningismus, serous meningitis, encephalitis lethargica, purulent meningitis and brain abscess obtained from an examination of the spinal fluid. The writer has never been able to do this although effort after effort has been made. In the present state of our knowledge, this is not feasible.

Special note should be made of cases of purulent meningitis, due to pneumococcus No. III, or what used to be described as streptococcus mucosus capsulatus cases. This organism, from the morphological standpoint, is a highly protected organism possessing a capsule 3 to 5 times the size of the organisms, and being protected against the attacks of the body fluids. It is of a low but persistent grade of virulence and once it invades the meninges is practically always fatal, although the clinical symptoms are so slight that the diagnosis of meningitis can only be made by an examination of the spinal fluid. The writer has seen a series of 14 of these cases, some under very

tragic circumstances, due to the surgeons not recognizing the organism they were dealing with. Hence, accuracy in bacteriological diagnosis is very important.

(2) Blood-counts. The blood-count in purulent meningitis is usually high. The leucocytes range from 25 to 50, and the differential often shows as high as 92 per cent. polynuclears. These observations do not apply to infections with No. III pneumococcus.

(3) Blood-cultures. In spite of recent reports, to the effect that purulent meningitis is often hematogenous in origin, the writer has only found two positive cases of bacteremia in the above series. The writer believes that the big majority of meningitis cases are direct extensions from the mastoids or sinuses and are not hematogenous in character.

Treatment

Here nothing much can be said since out of the above series only three survived. In one we used leucocytic extract and urotrofine intraspinously and the other two were left alone, as far as intraspinous treatment was concerned. The writer does not believe that any operative procedure will ever cure meningitis in any large percentage of cases, but rather that our endeavors should be toward the elaboration of antibodies to counteract the effects of the casual organisms, as in epidemic meningitis or the production of a chemical that will kill off the bacteria.

Brain Abscess

Very little of importance can be said about this condition. Out of a series of 74 abscesses, only 4 got well and remained well. The rest all succumbed in shorter or longer time. The writer feels that nothing could be gained by enlarging upon this subject as it has been so ably taken up by Dr. Dickie in his admirable thesis.

The sole purpose in writing the above article is to place the rather unique experience of the writer at the service of others in endeavoring to clear up some of the obscure and bothersome points that trouble us in the above condi-

tions, and if the experience outlined in this paper will be of assistance even once to any of the readers who have not had the opportunity of seeing such cases very often, then the writer's purpose will have been fulfilled.

Discussion

Dr. E. A. Crockett, Boston: This should have been a presessional paper, so that we could have had the full draft before discussion. I won't attempt to discuss the full subject, but would like to say that the speaker's remarks are particularly gratifying to me in regard to the conclusions on the ligation or exenteration of the infected vein. I, personally, since 1895, have been doing a routine operation on the lateral sinus, simple ligation of the jugular, no matter how large is the thrombosis or how serious the condition of the patient. This in our hands has proven a clinical success and I have never seen any reason for the exenteration of the infected vein. In the Massachusetts Eye and Ear, the statistics in uncomplicated cases without mastoiditis or symptoms of meningitis, the morality is not over 3 or 4 per cent, no matter whether the Chief of Staff or the house surgeon performs the operation. If there is meningitis or abscess of the lung, the mortality is high, these cases would be fatal after any operation. It is shutting the door after the horse is stolen. One point Dr. Dench brought out should be emphasized, the operator should never explore or handle an infected vessel without having ligated the vein, or thrombosis will be produced. In the cases seen at the Massachusetts Eye and Ear Infirmary, manipulation of the vessel produced thrombosis which did not previously exist. Dislodging of particles of the clot before the vessel was ligated was responsible for the symptoms. Another point of importance in the cases Dr. Dwyer spoke of last, as a matter of routine procedure, in injury to the sinus, the chances are the curet was full of pus and produced direct infection of the vessel without protective walling off. In all cases of injury to the sinus on

subsequent operation one should ligate the internal jugular. This should be a routine with younger men. That last case of Dr. Dwyer's would have been spared a fatal endocarditis. I always ligate as soon as the diagnosis is made where I have injured the vessel. I don't believe clinical pyemia always follows, but it does in a large proportion of the cases. We had a very conservative surgeon who had four cases of abscess of the lung following injury to the sinus because he refused to ligate. He thought the cases ought to get well spontaneously and he followed this idea to an illogical conclusion. Another point is that simple ligation is a quick operation, while exenteration is difficult. Much more time is consumed and there is much more shock to the patient with the general run of operators. I read a paper in 1907 on infection of the jugular. I ligated through a clot which was solid and not infected. In two cases there was complete recovery. After finding the clot it is a perfectly logical procedure to ligate it.

Dr. F. L. Jack, Boston: I am in accord with most of Dr. Crockett's remarks. In 1901 at the Third Pan-American Medical Congress, I reported two cases of ligature of the internal jugular vein and have employed this method ever since in infective thrombosis. I did not hear Dr. Dwyer mention what he did with the clot. Does he remove it? It is important.

Dr. J. F. McKernon, New York: My experience is somewhat at variance with the reader of the paper, and with those who have discussed it. I believe if you have an infected internal jugular vein, with infected clots, if you ligate it may go well if you have luck with you, but that is not my experience. This was borne out by a number of cases in France. In 14 reported cases ligated above the clavicle there were subsequent infections. Eight cases were operated upon a second time and the circulation cut off through the facial. The majority of cases did not get well. Dr. Crockett speaks of injury to the sinus. We

have all had these cases. In a few cases infection takes place. That is my experience in my own cases and in watching other operators. Ligation of the internal jugular is not enough when there is free pus in the sigmoid sinus. I do not believe it is good surgery to leave that in place. We must cut off the possibility of infection through the facial.

Dr. E. A. Crockett: We have 25 operators, 200 cases and 97 per cent recoveries. In the 14 cases the doctor mentioned and the patient showed elevation of temperature, I have seen such cases where the jugular was not ligated at all. Some small vein was ligated. There is the reason for variation in the statistics.

Dr. J. F. McKernon: In 8 cases I can testify that the ligature was round the vein. Fifty per cent died because of the empyemia following.

Dr. F. L. Jack: Was the vein opened above the ligature to see what was in the vein?

Dr. J. F. McKernon: The vein was opened at the time of operation.

Dr. F. L. Jack: I don't believe ligation is adequate without opening the vein to see if you have pus or clot. I don't excise clot. I leave free bleeding, but you cannot just ligate without removing septic material.

Dr. Edward B. Dench, New York: All roads lead to Rome, and a discussion of this kind is very profitable as it simply proves that a great many cases will get well, no matter what your procedure is. It proves also beyond a doubt that what one man calls sinus thrombosis sufficient for operation, another man does not. I am friendly to the Boston School, but I think that the jugular is tied in Boston on a single, or on one or two rises in temperature and this is considered a sufficient warrant to put a ligature round the vein. Very likely they are right. I do not believe,

however, that it is necessary in every case to ligate the internal jugular, although it may be safer, so that in New York excision is considered safer than ligation. I cannot agree that it is satisfactory to put a ligature round a thrombosed vein and to hope the case will get well. I had two cases where it was impossible to get a ligature round a healthy vein on account of the extent of the thrombus. Both cases died. I followed the vein down to the innominate vein. Both cases occured the same week. I think the doctors were very fortunate where they put a ligature round a vein with a clot and the patient did well. Many cases will get well with simple ligature where you ligate early. I cannot see any excuse for putting the ligature below the facial vein where there is every opportunity and every reason why the blood stream becomes infected from the facial and lingual vein because tonosus meet the opposite sides. I am very much against that procedure. I do not change my views on that point. As to the value of ligation as a first operation, the Boston School has convinced me. I do not think it is as simple as they say because there is always a mass of glands after point of election, but it is simpler than excision. When I think excision is always safe, but I believe ligation below the facial is never to be performed. In wounds of the sinus I believe ligation is reasonable. I had one case turning on this point. In a radical operation I wounded the lateral sinus. I had a blood culture made at the time of operation and it was negative. The man ran no temperature and the wound healed. On the 8th day after operation he had a temperature of 104°. At the end of 9 hours there were no colonies, at the end of 18 hours there were 12 colonies. I took out the vein and the temperature dropped and he made an uneventful recovery, so that was a clear demonstration that there was blood stream infection formed by sinus traumatism. If I had ligated the vein it would not have happened. My experience has been that of Dr. McKernon. I have seen many cases without general infection after wounding the sinus, but some cases get infected. I was doing a radical

operation some time ago, and found the sinus in the posterior tympanic space. As I curetted out granulations I got a stream of blood. The question arose, should I put a ligature round the jugular when the radical cavity had been completed. The field was clean. When I took out the plug which controlled the hemorrhage there was no bleeding, I argued the granulations must have been there for a long time. I put a clean graft over the whole vein and the patient left the hospital without any trouble. If you are certain of the field you can do this. With reference to the number of bacteria in the blood stream. The number may be very small and one does not get a positive culture. You may get a positive from the sinus but not from the median vein in the arm. In one case a culture direct from the sinus was positive. The temperature was 103°. In regard to the blood count, the differential blood count in meningitis is valuable. In sinus thrombosis there is about 12,000 leucocytes, the poly count is about 72-77, in meningitis it is about 95 per cent, while the leucocytes are as high as 20,000-30,000.

In atypical meningitis, particularly type III streptococcus capsulatus, I remember one case where the sinus was destroyed over the mastoid, the man was ready to go out. The house surgeon called me up and told me the man was in deep coma. He had streptococcus capsulatus in the spinal fluid, and died in 24 hours. I do not know any way of telling. You must do a lumbar puncture in all cases but if you find the organism you are powerless.

I do not agree with Dr. Dwyer. He says he does not care about the cell count any more than he does about pus in the wound. I care a whole lot about pus in the wound. When my cell count goes down I think the patient is doing better. The cell count is an index as to how the case is doing and also the relative severity of the symptoms. With a high cell count there is a Kernig and stiff neck and grave clinical picture. We also take note of the reduction of sugar and the globulin. With a negative Wassermann and chloride of gold, these points help to check up the case clinically.

Dr. J. K. M. Dickie, Ottawa, Canada: In sinus thrombosis ligature of the jugular vein alone is hardly enough. the vein should be opened and emptied, preferably above the common facial. If this is not done there is quite a danger of the blood in the vein forming a septic clot, which later breaks down into pus. I had a case of this sort quite recently in which I had to tie both ends of the divided vein as there was free bleeding from the upper end, evidently from the inferior petrosal sinus. The contents of the vessel became septic and I had eventually to open it up in its whole extent from the knee through the bulb to the neck before it cleaned up. With regard to spontaneous cure, I think that is possible. In another recent case I found the sinus filled with fibrous granulation tissue with no pus in the actual lumen although there was a large extra-dural abscess. The jugular vein above the common facial was collapsed and empty. This was a fairly good attempt of spontaneous cure. In Edinburgh the practice was to open the sinus widely, ligate and divide the jugular generally above the common facial leaving the upper end open for drainage.

Dr. B. Alex. Randall, Philadelphia: The harmony of our views reminds me of a musical experience in Philadelphia, when a lady, a niece of Benjamin Franklin, found fault with Theodore Thomas's Orchestra on account of 'instruments out of tune'. He said that it was the fault of 'the Philadelphia atmosphere'. She said 'Cericke with his Boston Symphony brings his atmosphere with him'. I think the Boston School has brought its atmosphere with it. Personally, I have not felt satisfied with merely tying the jugular: I have excised the vein. I think it is important in our teaching to consider this as phlebo-thrombosis of the lateral sinus. We have an inflammatory condition of the vein, whether of the meninges or the neck, and should not leave its septic material to be absorbed.

Dr. Ewing W. Day, Pittsburgh, Pa.: In regard to the symptoms of the meninges, we have been overlooking the

most important one, that is pressure. We hear the statement "the fluid is under pressure". The pressure within the cranial cavity is increased by irritation. In meningitis it is increased in proportion to the severity of the meningitis. I believe this is more important than the examination of the spinal fluid, except the bacteria, yet no records are being kept as to how the pressure is running. It is not necessary to waste much spinal fluid to get pressure. One c. m. of fluid is enough for every degree of pressure. I have diagnosed several cases on the pressure alone, where the fluid is negative so far as bacteria are concerned, but at post mortem you will get bacteria in the cerebrum. We can get a lot of information from the pressure alone. Many sinus thrombosis cases are never recognized, and go on to spontaneous cure. These are discovered at post-mortem, or in mastoid operations.

Dr. Emil Amberg, Detroit: There is another factor to be taken into consideration, and that is whether the jugular and the facial veins are the only ones which are liable to carry the infection.

Dr. S. MacCuen Smith, Philadelphia: Formerly it was a well recognized practice not to excise the vein in cases of sinus thrombosis where the clot had been removed and bleeding secured from both the torcular and jugular bulb ends. I should like to ask Dr. Dwyer if he feels that this is still a proper practice. Personally I have always felt it best to excise the vein, although on several occasions, owing to the extremity of the patient, after securing bleeding from above and below, I postponed further operative interference until the following day, and the patient then being much improved, a secondary operation was not done, all recovering without excision of the vein.

Dr. G. E. Shambaugh, Chicago: It is not always easy to determine in a particular case whether one has sufficient symptoms to justify an operation upon the sinus. Every

chill and a rise in temperature is not an indication of an operation on the sinus. Very often under just these circumstances an operation removing the infection about the sinu. but leaving the sinus undisturbed is sufficient.

A few minutes ago I raised the question of the proper procedure in cases of bilateral otitis media where nothing points more distinctly toward one side than toward the other as the source of the trouble, and where in the presence of chills with marked fluctuations in the temperature, one feels warranted in concluding that we are in the presence of a sinus thrombosis. But which ear is involved? Suppose one side is exposed. Are you going to ligate the vein then open the sinus and finding no evidence of thrombosis go through the similar procedure on the opposite side? I have been in the habit of exploring the sinus where after its exposure I cannot make out definitely whether it is thrombosed or not. The exploration is made with a good sized aspirating needle. Where both ears are involved with nothing to indicate which one is causing the serious trouble, if on aspirating, blood is drawn from the sinus I would proceed with the exposure of the opposite side and make a similar exploration. If the needle does not disclose an obliterating thrombus, I do not open either sinus. The one condition that may exist and not be detected by this method is a parietal thrombus. Experience has shown that a thrombus which has not undergone suppuration can usually take care of itself if left alone, provided the infection about the sinus is taken care of.

Dr. Edward B. Dench, New York: I consider parietal thrombi the most dangerous kind. The poison is brought into the circulation. In Dr. Shambaugh's case I would do double mastoid, and see if I could decide if both sinuses looked alike. I would think of the ear where the disease was most virulent or which had the most virulent organism and get free drainage. In a recent case, where the X-rays were very deceiving, I thought the patient might have a normal mastoid and I cut off the circulation on one side

and went into the mastoid. I happened to be fortunate in that case. Dr. Shambaugh's question is very appropriate. I saw one young lady who had a double tonsil and double antrum involvement. The doctor cleared up the antrum, then she developed double otitis media. Both ears were incised, then she had a temperature of 105°. The X-ray showed one side cloudy, one side not involved. One mastoid was opened and the jugular was tied. The temperature then went up to 106°. We thought of opening the other mastoid, but I thought of waiting before tying the other jugular. (If you ligate both internal jugulars do not obliterate both external jugulars, although the circulation can be carried on through the vertebrals). In this case as the patient came from S. America I thought of giving larger doses of quinine, and the temperature dropped and the patient recovered.

Dr. J. G. Dwyer (closing): I would never leave a clot in the sinus, neither would I tie off the jugular on one chill. In these cases the clinical and pathological symptoms didn't measure up, even where there was a partial clot. I wanted this paper to be as practical as possible so as to be a guide for the regular work. In New York we have to be shown, before tying off a jugular. All of you missed the point. You can get as much poison from the petrosal which you do not tie off as from the jugular which you do tie off. You leave the end open toward the bulb; the bulb is cureted out and pressure is equalized. The weakest point is in the bulb, and you can get out liquid pus through the bulb. Where your organisms go in you get a coagulation necrosis, then you get liquefaction necrosis. Where you have inexpert assistance, ligation is the easier operation. I do not think it is necessary to ligate immediately in clots of the sinus. In case of injury to the sinus with rise of temperature, one should tie off. Dr. Dickie says he excises the jugular and leaves the end open. What does Dr. Dench do? You can also get bleeding from the petrosal. We have been using the manometer, but I

do not think it is a question of pressure. The greatest pressure cases were from intoxication of the central nervous system. One case had high delirium. We ligated the jugular, and the patient became normal. The question Dr. Shambaugh raised depends upon whether the glands are involved upon one side. I have seen a double mastoid in a boy of 10, with distinct tenderness over the jugular on one side. We operated on that side and the boy got well. In another case there was a board-like feeling with the pulse transmitted to the carotid. I always say a prayer over the double cases. It is bad surgery to interfere with the sinus before you touch the jugular. If you have a mastoid and sinus thrombosis, with positive blood culture, stop the work there and tie the jugular. Then you can open the sinus safely.

SKIAGRAPHIC STUDIES IN EAR DISEASE WITH RELATION TO THE DEGREE OF PNEUMATIZATION OF THE MASTOID.

By NORVAL H. PIERCE, M. D., Chicago, Ill.

The principle purpose of these skiagraphic studies is to ascertain the condition of the mastoid as regards pneumatization of the mastoid in a series of cases of hardness of hearing due to disease of the sound conducting apparatus other than those with chronic suppuration.

The diagnosis was made by the usual functional tests, the Bezold-Edlemann continuous tone series being used, together with the Bezold large A for the Weber-Schwabach, (normal bone conduction 33 seconds), a small a^1 for the Rinné (normal bone conduction 16 seconds, aerial conduction 40 seconds), the Lucae c^4 for upper intensity and the Struychen's monochord for the upper tone limit. In making the Rinné test both the duration of bone conduction and aerial audibility were recorded in seconds, a procedure which should always be carried out if we are to gain the full value of the test.

The first skiagraph is, perhaps not intimately connected with the subject as it is a case in which pneumatization is normal, but it exemplifies a class of cases in which the principle features are increasing hardness of hearing due to stapes anchylosis with otoscopic evidence of caval inflammation.

A. M., female, aged 37, complains of hardness of hearing in the left ear with a high-pitched, ringing tinnitus. Cause unknown. Has noticed the defect of hearing for four years: it does not seem to be progressing lately. Complains of sensation of light headedness, but the description is not that of true vertigo of vestibular origin, and there is no nausea. Never had a discharge from the ears. Father is hard of hearing. Had measles when a child.

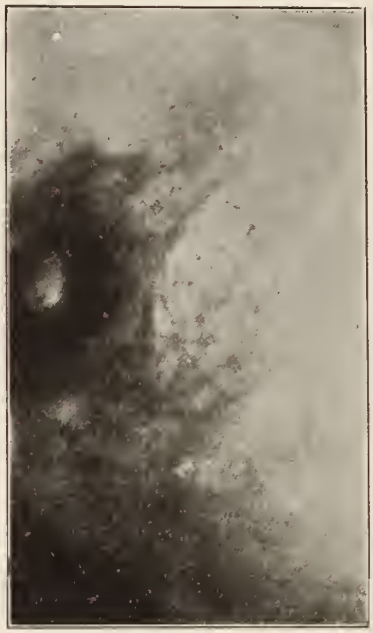
Functional Tests: A. D. Whisper, Numerals 30 feet, medium pitched words 18 ft., Weber not lateralized 23 seconds. Rinné positive, (A. C. 35 seconds, O. C. 14 seconds). Low limit G2, 24 D. V.)). High limit B^6. A. S. whisper. High and medium pitched words 5 to 6 inches, low pitched 2 inches. Rinné, negative, (A. C. 12 seconds, O. C. 15). Low limit, A1 (55 d. v.). High limit

Case I—Right.

A^5, C^4 very much shortened. Turning to right, nystagmus, 8 seconds, vertigo 15 seconds. Turning to left nystagmus 10 seconds, vertigo 20 seconds. The right tympanic membrane shows a light reflex reduced in length, lusterless, darkened and the handle of the malleus is situated nearer the posterior periphery than is normal. The left mt. is slightly more retracted than the right, the light reflex is absent, the color is darker, it is lusterless and the handle

of the malleus is nearer the posterior periphery than normal. No improvement on inflation.

This is an instructive case. Obviously a disease of the left sound-conducting apparatus, (fixation of the stapes) as witness loss of the low limit and negative Rinné with involvement of the sound perceiving apparatus as witness of loss in normal bone conduction and the loss in the upper



Case I—Left.

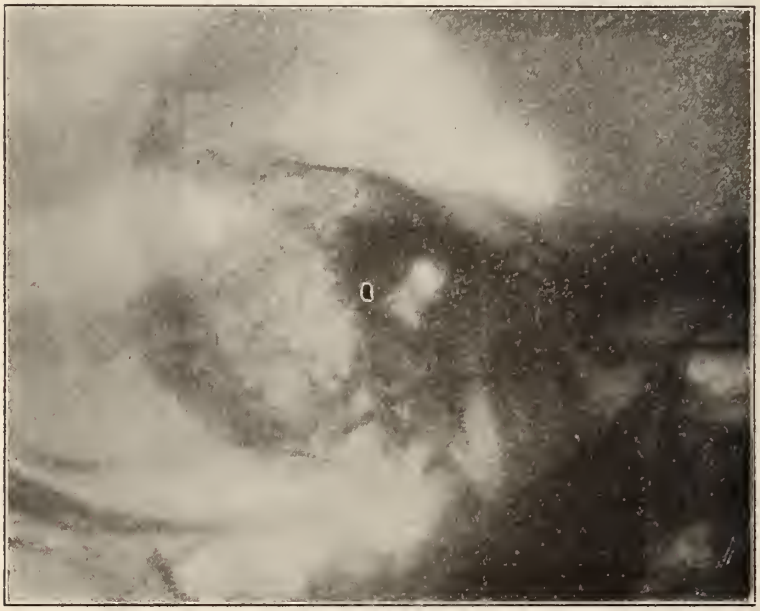
tone limit on the left side. The appearance of the tympanic membranes was indicative of the presence in the cavum of a plastic (catarrhal) inflammatory process. The position of the handle of the mallei is frequently seen in cases of arrested pneumatization. It is interesting to see by the skiagraph how extensively pneumaticized the mastoids are. In view of the history of deafness in the family of the patient we are inclined to regard the fixation of the stapes

as due to oto-spongiosis, and not to the inflammatory processes to which the hyperplastic mucous membrane which accompanies arrested pneumatization is subject. The appearance of the mt. indicates that those tubo-tympanal in flammatory processes may occur in connection with normal pneumatization. However, the Roentgenogram aids materially in forwarding the diagnosis and prognosis.

Case II—Right.

The second Roentgenograms are from the mastoids of a female child, 8 years of age. They show complete (normal) pneumatization. This child has been under my care since she was thirty-two months old at which time she had an acute suppurative otitis media, which ran a normal course and ended in recovery, although there was marked tenderness and some infiltration over the mastoid fossa in the first days of the disease. Since then she has had two

additional attacks of acute otitis media, but of much less severity. Yet we find that although these three attacks of acute otitis media occurred during the period when the process of pneumatization of the mastoid was most active they in no way interfered with its progress on to extensive normal development. This is in accordance with the conclusions of Wittmaack. So long as pneumatization



Case II—Left.

is not interfered with before the beginning of the second year of life pneumatization proceeds. That which causes arrest of pneumatization must occur in the first year, and that which occurs is not an acute inflammation, but a chronic latent process—the otitis media neonatorum. This is an example of a series of similar cases.

The third Roentgenograms are from a female patient, (A. H. 33-B-7), aged 22, who complained of recurrent

attacks of earache and discharge since childhood with increasing hardness of hearing. The right tympanic membranes showed a diffuse light reflex with fluid back of it in the cavum. No perforation. The left membrane showed areas of opacity and atrophy with injection of blood vessels along the malleus, no perforation. The whispered voice was heard in the right ear 10 ft., for low pitched words,

Case III—Right.

18 ft., high pitched words, left ear low pitched words 6 inches; high pitched words 12 feet. Weber-Schwabach to the left 33 seconds. Low limit right ear D^1 (36 D. V.); left ear D^1, Rinné right ear o. c. 18; a. c. 27=+9 seconds. Left ear o. c. 26; a. c. 18=-8. High limit right ear a^6 left ear f^6. Inflation brought hearing for whispered voice in both ears to over 20 ft., for both high and low pitched words. The Roentgenograms show marked arrest of

pneumatization in the right and total arrest in the left mastoid. Evidently the fixation of the stapes was not permanent. Nose, post-nasal space and pharynx negative.

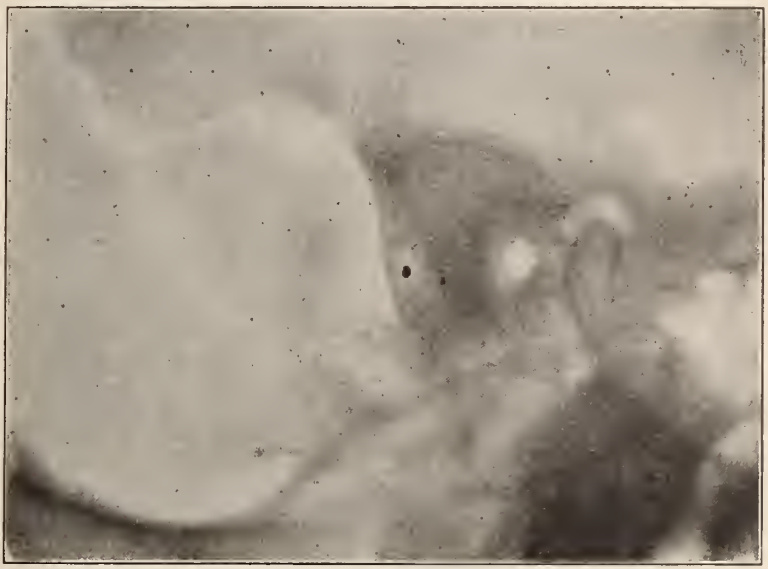
This case is typical of a large series which give a history of recurrent attacks of acute otitis media with discharge occuring since childhood, the discharge being purulent or, more frequently, mucoid or serous. The attacks in these

Case III—Left.

cases are of short duration, but the hearing is most often increasingly impaired.

The fourth Roentgenogram is from a female (E. M. 3-b), aged 64 years, with hardness of hearing in the left ear for 10 years. The cause unknown. There is slight pain recently, a crackling tinnitus in both ears. Her brother was hard of hearing. Never had a discharge. Membrana tympani right: areas of thickening (opacity), the light

reflex is distorted and reduced in length and breadth. No perforation. Left: atrophic, light reflex is distorted and displaced. No perforation. Functional tests: Whisper, right ear 20 ft.,+. Left ear: low pitched words 10 ft., high pitched words 12 ft., Rinné right o. c. 11 ft., a. c. 25 ft., =+16; left ear o. c. 11 ft., a. c. 23 ft.,=+12. Low limit right G-2, left D-1 (36 d. v.). Weber-Schwabach unlateralized 32 seconds. High limit g^6 in both ears. After



Case IV—Right.

inflation the hearing for whisper in the left ear came up to 20 ft., for high pitched words and low and medium pitched words 15 ft. Nose, postnasal spaces and pharynx negative.

The Roentgenograms show the right mastoid to be unpneumaticized in the posterior portion, only a few isolated cells showing in the region of the antrum, while the lower portion is diploetic. The left shows the same picture, except that the cells are fewer in number and are more obscure.

This is a case in a large series of tubo-tympanal catarrh. From the appearance of the Roentgenograms we must conclude, in the light of Wittmaack's work, that the mucosa is thickened and that the cavum is the seat of those pathological changes which result from otitia media neonatorum.

I will not take up your time by exhibiting more pictures.

Case IV—Left.

Those presented represent many which have been taken in the past three years. The skiagraphic study of some hundreds of plates strengthens the opinion that Wittmaack's conclusions are in the main correct. We invariably find arrested pneumatization in chronic suppurative cases. But as you have seen in those cases which present the picture of what we have called chronic catarrh of the middle ear and tubo-tympanic catarrh with no history of chronic

discharge we also find arrested pneumatization in various degrees. Nor, is it a rarity to find more or less asymmetry between the two sides. The less pneumatized corresponding usually to the more affected ear as determined by functional tests.

For the purpose of description we may divide the mastoid into three types as shown by the Roentgen ray.

1. The completely (normal) pneumaticized mastoid. That is a mastoid in which the cortex is thin and the cells occupy the whole mastoid process to the tip, backwards and upwards to the occipital suture or further and above the antrum and cavum toward the zygomatic root. The cells are round and are systematically arranged with the larger cells terminal. With such a picture we may know that the mucosa lining the cavum and cells is normal—that is the epithelium cell is epithelioid—very thin and consisting of one or two layers. The subepithelial tissue is also very thin, homogenous, and cannot be separated into layers.

2. The mastoid in which pneumatization has been completely arrested. Here we may know that the mucosa in the cavum and antrum is thick, the epithelium cuboid with areas of ciliated epithelium, the subepithelial tissue is also thick and rather sodden, it may be separated into several layers, one of which may be identified as consisting of embryonic myxomatous tissue. It is richer in blood vessels and this is infiltrated by round cells. In the cavum and antrum are abnormal strands and sheets of tissue, cysts and organized deposits.

3. Mastoids in which pneumatization is partially arrested. These may be divided into:

a. Those in which we find the cells present only at the upper and posterior periphery of the antrum.

b. Those in which the cells are present in this region and have spread downward between the external auditory canal and the sinus.

c. Those in which pneumatization has proceeded so that the cells cover the sinus, leaving the posterior portion

of the mastoid solid.

In all of these we have a thickened membrane such as we sketched under type 2, of fibrosis which may have taken place in the thickened membrane, in which case the terminating line of the cell will be abrupt. In order to appreciate the reason why the ascertainment of the condition of the mastoid as regards pneumatization is of scientific interest in this series of cases of non-suppurative disease of the sound conducting mechanism, a knowledge of the work that has been done in recent years on normal and abnormal pneumatization of the temporal bone is necessary. To those who are unacquainted with this work or to those whose memory of it has become dimmed, a brief outline of it needs no excuse.

Up to the recent past three types of mastoids were regarded as normal, id est[1], the wholly pneumaticized mastoid[2], the partially pneumaticized mastoid and the[3] sclerosed mastoids, or mastoids in which air cells were absent. Arthur Cheatle was the first to hint that the second variety was due to developmental irregularity and referred to these mastoids as of "an infantile type." The third type, the so-called "sclerosed' mastoid was believed to be frequently due to sclerosing process, the results of chronic suppuration, having obliterated previously existing pneumatic spaces. Wittmaack was the first to study the mastoid histologically in serial sections and in 1918, (?) published the results of his work of eleven years on normal and abnormal pneumatization of the temporal bone.

He divides the normal pneumatization of the temporal bone into three stages. In the first period we have the formation of the cavum, recessus and antrum. At birth these cavities are filled with myxomatous tissue. At first (5th fetal month) there is a mere slit existing between this myxomatous tissue and the tympanic membrane. The myxomatous tissue is covered with cuboid epithelium with its face to the tympanic membrane. Gradually the embryonal tissue is absorbed or disappears and the epithelium following it lines the air-containing cavities; first the

cavum, then the recessus and at last the antrum. Normally, when this pneumatization is undisturbed, the submucous tissue consists eventually of a very thin layer over the bone and the epithelium changes from the cuboid type, normal in the embryo, to the endothelioid epithelium, one or two cells in thickness.

At the end of the first or beginning of the second year the second stage begins and consists in the entrance of myxomatous tissue from the antrum into the marrow spaces of the underlying spongiosa. The marrow cells of these cavities are absorbed and replaced by the myxomatous tissue and at a given time the epithelium from the antrum dips down and as it dips down the myxomatous tissue is in turn regressed until it becomes the submucous layer with the epithelium covering it. Thus an air space is made. This process goes on continuously by the invasion of narrow spaces by the myxomatous tissue from previously formed cells until consecutively the air cells are completed at the end of the fourth or beginning of the fifth year.

The third stage extends through life and is denominated by Wittmaack as the stage of interstitial pneumatization. This consists of the formation of small pneumatic spaces communicating with the larger spaces which were formed in early childhood in the nodal points of the network which forms the walls of the pre-existing pneumatic spaces.

His conclusions are, (1) the development of pneumatic systems in the temporal bone, that is, the tubal cells, the tympanic cells and the mastoid cells follow a more fixed process of development with far greater regularity than has heretofore been believed. (2) The normal structure of the pneumatic process is characterized by a far-reaching pneumatization and not by a sclerosing process. The individual peculiarities in the formation of the pneumatic system in later life, that is, after the fifth year, is not manifested by the peculiarity of the mastoid as a whole, but only as an interference in the peripheral structure of the pneumatic system. It will be seen, therefore, that he

recognizes but one type of normal mastoid and that is one in which complete pneumatization occurs. The presence of spongiosa is a sign of arrested development.

What is the cause of this arrested development? And here perhaps we have the corner stone of Wittmaack's deductions. In seeking for the cause of this disturbance of pneumatization he reasons that the cause must primarily be searched for in the middle ear mucosa, the osseous structures evidently playing a secondary part in the process of pneumatization. Even in the normal process it is quite evident that the bone is subordinate to the changes which are wrought in it by the submucous myxomatous tissue. It has been known for a long time and written about by many authors that the middle ear of the newborn and sucklings is subject to a peculiar inflammatory process which has been denominated the otitis media of the newborn, otitis media concomitans, etc.

Schwartz, (Archiv. fur Ohen., Band 1, page 204) in 1864 recognized the "enormous frequency" with which inflammatory conditions are found post mortem in the middle ear in infancy and in the description of the findings in one of his cases he says: "the middle ear is full of a thick, greenish-yellow pus which contains well preserved ciliated epithelium. The covering of the middle ear is of a deep dark red color and is noticably loosened. * * * **The ossicles are completely embedded in the swollen mucous membrane.*" There was no perforation in the tympanic membranes reported. This description tallies closely with that of Wittmaack in describing the same condition as it occurred in his material. If I may digress for a moment I would draw attention to the condition Schartze found in the inner ear of these cases as it throws a very suggestive light on the frequency with which we discover involvement of the sound-perceiving apparatus in cases of arrested pneumatization in later life.

Von Troltsch also recognized the frequency with which otitis media occurred in infancy and young childhood. In a series of 24 post mortems on children he found the condition in 17.

However, Schwartze yields the palm of priority for recognizing the frequent occurrence of what we now call otitis media neonatorum to du Verney who is known for the numerossity of his ear sections. In du Verney's "Tractus de Organo Auditus" (1684) page 36, he says: "Aperui etiam complurium infantium aures, in quibus tympanum excrementis erat plenum, interim numquam, neque in cerebro, neque in osse petroso, inventa ulla prava dispositione."

It is latent in its character and course and is discovered most frequently at postmortem table. According to Solowzow, over 90 per cent. of all newborn children and infants are subject to this inflammatory process in the middle ear. Preysing, Görke, Rietschel, and more lately, Göffert have extensively investigated this form of otitis media. The frequency with which it is found in children or infants rendered it difficult for Wittmaack to secure a large number of undoubtedly normal temporal bones in the first year of life. Much more easily could temporal bones with this inflammatory process of the mucous membrane be secured. He concludes that the process is not physiological but is undoubtedly a pathological condition, and this view is supported by the presence of pus in the cavities of the middle ear and the known anatomical changes in the mucosa. These anatomical changes have been especially studied by Görke and Rietschel and they agree that there are two definite types, a plastic and an exudative form, and Wittmaack suggests as a result of his investigations that these may be divided into a latent insidious type and a more acute form with a relatively rapid course. To these forms Wittmaack adds the hyperplastic changes in the plastic otitis media and an atrophic form with an exudate. It is evident that in the majority of cases we have a mixed type of these pathological changes. The pure type of the first group is, however, clinically without symptoms and is, therefore, unsuspected during life. How this can affect the pneumatization will appear later.

The cause of this latent, symptomless, insidious form of inflammation was ascribed first by Aschoff to the aspiration of amniotic liquor, vernix or meconium into the tube and then into the cavum. The entrance of this irritant may occur in fetal life (aspiration of amniotic liquor), or during the time of birth (aspiration of amniotic liquor, vernix and meconium). The pollution of the cavum with amniotic liquor is, according to Aschoff, the cause of the presence of leukocytes in the cavum of the newborn. According to this view otitis media neonatorum is not an infectious process but rather a reaction to a foreign body. It does, however, according to Hartmann and other authors, render the structures more disposed to bacterial invasion. This is in agreement with the frequency with which bacteria are found in the exudate of the middle ear of newborn children. However, the foreign body reaction with sterile exudate may effect changes in the mucosa which will interfere with or check pneumatization. In fact, Preysing in a great number of all ears which he examined in the newborn found an exudate which was sterile. Among the infected cases the pneumococcus was by far the most frequently found. The changes wrought by the inflammatory process in the mucosa are characterized by a more or less intensive infiltration of the superficial layers of the mucosa and the epithelium with round cells, dilatation of the blood vessels and production of an exudate containing more or less pus corpuscles. The epithelial strata is changed so that the ciliated epithelium (lining the tube) is found in places far removed, in the recessus and even in the antrum, areas in which the ciliated epithelium is never found in normal conditions. On this point Wittmaack differs with Preysing and Görke. His conclusion, based on a study of a large amount of material is that the extension of ciliated epithelium over the mucosa of the middle ear must be regarded as a pathological process. He has proven by experiments on the lower animals —rabbits, cats, etc.,—by production of a purulent inflammation in the ear that he can produce a spreading of ciliated epithelium.

The normal epithelium of late fetal or early infantile life in the cavum is cuboidal in character which changes under normal conditions to the epithelioid type. The persistance of the cubodial epithelium and the presence of ciliated epithelium Wittmaack regards as a mark of pathological changes. The persistence of the cuboid and ciliated epithelium coincides with other evidences of pathological conditions in specimens showing interference with pneumatization, to a degree that suggests that his views are correct. Moreover, these abnormal epithelial types are not present in normal pneumatization.

Allowing for a relatively broad individual latitude in normal regression of the submucous tissues, there comes a time in the third to the fourth month where a normal type may be differentiated from an abnormal type. There can be no doubt that in the presence of the inflammatory process under consideration the superficial layers of the mucosa and the deep subepithelial myxomatous tissue shows no tendency to regression but, on the contrary, displays a tendency to thicken. Wittmaack differs from Preysing in this regard and criticizes one of his illustrations which Preysing displays as a normal mucosa as being evidently an abnormal condition dependent upon an inflammatory process. Wittmaack believes that a normally developed mucosa cannot be differentiated into various layers and when this is possible it is due to disease.

We have, then, the persistence, more or less complete, of the embryonal subepithelial myxomatous tissue, but undoubtedly a proliferation of the same. We have then an exquisitely hyperplastic mucosa rich in blood vessels. The superficial layer under the epithelium shows marked infiltration with round cells. Also there is a typical formation of lymph follicles. This thickening of the subepithelial tissue is especially to be found in the niches of the cavum and recessus. Another change which is characteristic of this condition is the formation of the granulation nodules.

The point is made by Wittmaack that this inflammatory process causes the total or partial arrest of the recession

or disappearance of the myxomatous embryonal tissue. This does not occur in a regular manner over the entire surface, but in a very irregular manner depending largely upon the inflammatory intensity at a given point. It can be readily understood how bridges of membrane may thus be formed by an incomplete resorption, or abnormal strands of tissue, can arise especially in the recessus epitympanicus and about the foot of the stapes—anywhere where there are sharp corners and depressions—by residual tissue.

Granting that the inflammatory process causes persistence of the thick subepithelial portion of the mucosa represented largely by a hyperplastic type derived from the former myxomatous tissue, and remembering the manner in which the epithelium follows the myxomatous tissue into the marrow spaces of the bone it can be readily understood how the process of pneumatization is arrested. It seems that the subepithelial tissue is deprived of its power of invading the marrow spaces and where this does occur in a limited manner the epithelium has no power of penetrating, but remains as a level layer on the thickened submucous tissue.

He concludes, first, divergences from the normal structure of the mastoid depend, without exception, upon typical processes in consequence of changes in the character of the mucosa in the first and second years of life.

Second, the changes in the character of the mucosa may be grouped under the hyperplastic and fibrous types.

Third, the hyperpastic type develops from a latent insidious plastic inflammatory process in the mucosa.

Fourth, the fibrous type depends on an acute exudative inflammatory process.

Fifth, whether disturbance of the pneumatization is partial or complete depends upon the intensity of the changes in the mucosa.

Sixth, every type of disturbance of pneumatization gives a typical structure picture of the mastoid—

I. Complete arrest of pneumatization.
   a. By hyperplastic.
   b. By fibrous mucosa.

II. Partial arrest of pneumatization.

a. In the hyperplastic inflammation (severe, intermediate and light grade).

b. By fibrous mucosa (prolonged pneumatization).

Seventh, the concurrence of hyperplastic with fibrous changes in the mucosa occur with relative frequency and lead to mixed forms of structure types, sometimes with one and sometimes with the other component predominating.

What relationship does pathological pneumatization bear to changes in the tympanic membrane? As the tympanic membrane is the one visible portion of the otic structures, it is interesting and important to know whether the changes in this membrane bear any relationship to pathologic pneumatization. At the present time it is difficult to say just what relationship slight and moderate anomalies in the tympanic membrane bear to the pneumatization process. This is readily understood when we remember that changes occur rapidly in the tympanic membrane after death. Farther, the separation of the tympanic membrane from the caval myxomatous tissue occurs in mid-fetal life,—before the 5th month. Wittmaack believes however, that he is safe in saying that the manubrium in the otoscopic examination lies in a course that is nearer to the posterior external auditory canal than in normal cases and the circumference of the tympanic membrane appears more oval and is smaller than normal in cases of disturbance of pneumatization. In other words, the infantile type of tympanic membrane persists. Changes in the tympanic membranes themselves may or may not have a relationship to the character of the mucosa within the cavum. He points out, however, that slight diffuse and regular cloudiness of the tympanic membrane with a diminution in the luster is an indication of disturbance of pneumatization, together with the other changes in the mucosa which accompanies these disturbances. This cloudiness is due to increase in the cuticle layer of the tympanic membrane and the decreased luster is due to a greater desqua-

mation of the superficial layers. The cuticle layer of an entirely normal adult consists of a single layer of perfectly flat epithelium and this gives a bright luster. In hyperplastic changes of the mucosa we often find the deep cuticle layers composed of cuboid cells. The subepithelial layers show a thicker and richer vascularity. The superficial layers of epithelium are raised in lamella, which give them a rougher surface. This gives the cloudiness and lack of luster of the tympanic membrane on otoscopy. This is a normal condition in the first period of development and reaches frequently into the second period. In this way it is explained why in sucklings and small children we seldom find a tympanic membrane with the luster of the adult tympanic membrane. We very frequently find by otoscopy atrophy of the tympanic membrane in connection with fibrous changes in the mucous membrane with complete or marked arrest of pneumatization. This is due to the arrest of the membrana substantia propria.

Abnormalities in the tympanic membrane are associated with arrested pneumatization indicated by spotting of the tympanic membrane by areas of thickening associated with areas of atrophy; the areas of thickening appearing as whitened islands, while the areas of atrophy give a darker tone due to their translucence and the color of the membrane within the tympanic cavity.

Calcareous deposits in the tympanic membrane are not associated with changes in pneumatization. Peripheral cloudiness of the tympanic membrane is frequently associated with changes in the mucosa accompanying pathological pneumatization.

What is the relation of pathologic pneumatization to the accessory mucous membrane bands and folds? It is known than in the recessus epitympanicus is found a number of ligaments and duplicatures of the mucosa. Wittmaack attempts to answer the question: What is the absolutely normal anatomical content of the epitympanic space? If we confine ourselves to the temporal bone with a completely undisturbed development we can say that in the

recessus we find besides the two folds, the ligamentum malei externum sive laterale, the ligamentum malei superior and the ligamentum malei anticum. Occasionally one or the other of these ligaments is lacking. Especially is the ligamentum malei superior inconstant. It is absent with relative frequency in high tegmens and well developed pneumatic cells.

When the bones are examined at a time when the myxomatous tissue is not yet completely absorbed the anlage of these ligaments can be discerned within the tissue in the form of fibrillary strands and presents a resistance to the ingrowing epithelium similar to that displayed by the ossicles.

The development of the inconstant so-called accessory folds frequently occurs from the incomplete absorption of the subepithelial tissue. In this way small bridges of tissue or sheets of tissue originates, reaching from one wall to the other of the epitympanic space. Zuckerkandl has previously described the development of accessory folds in a similar manner.

The arrest of pneumatization in the mastoid has a tendency to draw the sigmoid sinus forward, a very practical fact to remember in our operations on such cases. Especially noteworthy, is the protocol in one of his cases (No. 38) in which complete arrest of pneumatization is pictured, and where the displacement forward of the sigmoid sinus is most marked.

In summing up the relationship which arrested pneumatization bears to other parts of the temporal bone we find:

1. There undoubtedly exists a certain relationship between pathological pneumatization and certain anomalies of the tympanic membrane, lusterlessness, cloudiness, atrophies, scars, etc.

2. Changes in the tympanic membrane cannot be regarded as always constant accompaniments of pathological pneumatization.

3. In entirely normal pneumatized temporal bones we find only the constant ligaments in the epitympanic space.

The development of accessory folds is a sign of pathological pneumatization.

4. The displacement forward of the sigmoid sinus is found in pathological pneumatization. The higher grades of displacement only with the worst form of disturbance of pneumatization.

5. The persistance and unusual breadth of the fissures is an accompaniment of pathological pneumatization.

It is apparent that the work of Wittmaack explains many hitherto unexplained problems. It explains, for instance, the so-called chronic catarrhal otitis media which arises from apparently no cause and which has been explained on the hypothesis of diathesis or a catarrhal inflammation. It explains the chronic tubo-tympanal inflammation, or at least places these conditions in an entirely new light.

The more important conclusions as regard the relation of arrested pneumatization to inflammatory disease of the ear may be summed up as follows:

I. Practically all severe forms of suppurative middle ear involvement develop in temporal bones with pathological pneumatization insofar as this depends on continuous extension from the tube.

II. Chronic middle ear suppuration exists on the basis of complete arrested pneumatization, or the severest forms of disturbance of pneumatization, with markedly hyperplastic mucosa and develops in the child;

A. Chronic suppuration of the mucosa on the ground of the acute exacerbations of the suckling.

B. The chronic cholesteatoma suppuration, either (a) after acute necrosing otitis through ingrowth of the epithelium in consequence of large peripheral defects of the tympanic membrane, or (b) as an insidious process with intact of the pars tensa after sequestration of the antrum recessus from the cavo-tubal cavity in consequence of adhesions from Schrapnell's membrane, or an atrophy above, or adhesions above or below the posterior folds; (c) middle ear suppuration with epithelization or combination recessus cavum and cavum cholesteatoma suppuration

and a combination of the process which leads to cholesteatoma formation.

III. The form which the chronic suppuration and its course pursues is preordained by the anatomical changes within the several cavities of the middle ear before the appearance of the clinical symptoms. Also, the secondary and end processes, such as the extent of perforation, epidermization, polyp formation, scarring, etc., depend upon a preformed anatomical substratum.

IV. The acute middle ear suppurations develop with predilection in medium and lighter grades of disturbance of mucosa and corresponding character of the mucous membrane.

V. The greater the hyperplasia, the thicker the epithelium, and the less the pneumatization, the greater is the tendency toward the occurrence of acute inflammatory process and eventually a protracted course, and the less, on the contrary, is the tendency to frank mastoiditis and vice versa.

VI. The character of the secretion in an acute otitis media stands in direct relationship to the character of the mucosa. Thick, highly hyperplastic mucosa with ciliated epithelium is especially apt to produce a mucous or mucopurulent secretion. Slight hyperplasia with flat epithelium gives thick, purulent, tenacious, secretions. Fibrous changes predispose to a thin fluid, serous or seropurulent secretion.

VII. Normally pneumatized temporal bones are most frequently infected in acute middle ear disease with resulting frank mastoiditis.

VIII. Middle ear inflammations of tubercular and leutic character exist on the anatomical substrata of the mucous membrane and its accompanying disturbance of pneumatization. Their course depends also on the changes in character of the mucosa as it occurs in the course of pneumatization.

The relation which pathological pneumatization bears to endocranial complications is most interesting and impor-

tant. In the pictures which I shall cause to be thrown on the screen you will find how frequently abnormal vascular communications persist between the abnormal mucosa, the meninges, the bulb of the jugular and the sigmoid sinus.

It would be impossible to give a complete account of the monumental work performed by Wittmaack. We must be content with this short and incomplete sketch.

Before closing I must, however, accentuate the very kernel of Wittmaack's deductions, namely, that nearly all inflammatory diseases of the middle ear, in their genesis, nature and course are dependent in certain anatomico-developmental changes in structure of the mucosa and osseous structures of the ear. Most of these alterations in structure are caused by a latent, insidious, inflammatory process which occurs in early life. In other words, if in late life an individual develops middle ear inflammation (catarrhal) with adhesions, fixation of the stapes, etc., the special suppurative type of inflammation, is predestined, when the occasional cause arrives, by the changes which have occurred in the first years of that individual's infancy. On this fundamental principal he has erected a plausible, logical structure which must be proved or disproved by future investigations. True, there are discrepancies; here and there we discover findings and conclusions which are susceptible to quite different interpretations, but, on the other hand, he throws light on many marked dark corners of otology and explains in a logical manner many points of pathogenesis which have heretofore been merely surrounded with meaningless words.

Discussion.

Dr. Edward B. Dench, New York: Two of the plates were interesting as showing to what an extent pneumatization of the bone can be carried.

Dr. B. Alex. Randall, Philadelphia, Pa.: I am unable to accept the views of Wittmaack. I have studied his work

with great care and am utterly unconvinced by his conclusions, as I feel that a great many points shown by his pictures could be interpreted differently and that a good many deductions that he draws are unjustified. In making corrosion casts of the temporal bones 30 years ago, I found one case where the softening of the bone in the potash solution gave me only two chains of metal-filled cells. These were complete, reaching to the tip in both directions. The rest of the bone showed the septa or walls of a series of cells which contained ivory-like nodules representing the calcified mucosa and easily separable as the pulps from the skins of bunches of grapes. We have often seen cases where 'granulomata' can form in the cells in one day. The antrum of Highmore can be filled up with swollen edematous mucous membrane in one day and form 'granuloma', which disappears as quickly as it forms. In the inflammatory process the mucosa has undergone eburnation and the process has completely filled the cavities of the mastoid. This convinced me that Buck and other clinicians are right in claiming that these are cases of eburnization taking place from chronic inflammation.

Dr. Francis P. Emerson, Boston: I was interested in Dr. Pierce's pictures and also in Dr. Randall's comment. I went over the records of the Massachusetts Charitable Eye and Ear Infirmary for the past six months and found that we have a record of 341 mastoid operations, with 41 cases of arrested pneumatization or a sclerosed process which had taken place in a pneumatic mastoid. I think we can distinguish the infantile type of mastoid from the position of the sinus. If the sinus is far forward, it shows arrested development. If it is far back it is the fully developed adult type. We had three cases of particular interest because the infection was of a low-grade character. The patients came in with middle ear symptoms and mastoid tenderness which continued about three weeks without breaking down. In these three cases, so far as one could judge by the X-ray, there was a gradual sclerosing proces-

going on. In the infantile type, Dr. Pierce's explanation of the way meningeal symptoms take place is interesting but I think in the infantile type we should remember that we get cholesteatoma and pressure symptoms with danger from that source. The speculation of Wittmaack is a very large subject and one that will require a great deal of study and observation before we can come to a definite conclusion, especially in view of the increasing number of cases showing accessory sinus involvement in early years..

Dr. Emil Amberg, Detroit: One point should be brought out: that is the value of the X-ray in the diagnosis of middle ear conditions. Wittmaack mentions the assistance of the Roentgen-ray picture in the diagnosis of oto-sclerosis. Oto-sclerosis develops, according to Wittmaack, contrary to the opinion of Katz, independently of the changes of the mucosa of the middle ear. We can thus draw conclusions from the X-ray picture, as it reveals the character of pneumatization. Under certain circumstances, good pneumatization may be of diagnostic aid. Wittmaack published a book on oto-sclerosis in 1919 . He claims the mucosa has nothing to do with oto-sclerosis. I believe the X-ray will throw light on suppurative processes as well as on oto-sclerosis.

Dr. Norval H. Pierce, Chicago, (closing): I brought pictures to show that there is arrest of pneumatization in many cases of non-suppurative (catarrhal) otitis media. We formerly thought obliteration of the pneumatic spaces of the mastoid was due to suppurative inflammatory changes in the bone. In the majority of the cases I have shown there never was a chronic discharge from the ear. The drum membranes were intact. On account of the poor showing of the plates I could not point out that there is often asymmetry, as regards pneumatization, between the two sides. That means that in a large series of cases of patients coming for advice in connection with so-called catarrhal deafness, we have found considerable arrest of

pneumatization. It was too bad that Dr. Randall could not have preserved that speciman from which those little spheres were taken. I do not see how they developed in pneumatic spaces, or how they became disconnected from the walls if they did form.

We must bear in mind that there are many degrees of disturbance of pneumanitization, from very slight arrest to complete arrest. In cases where the arrest is limited to the posterior sinus, we may have involvement of the anterior series in acute suppuration. These are the dangerous cases. They suddenly develop sinus thrombosis without tenderness or swelling outside because of the enormous thickness of the bone and the abnormal vascular supply. These are the cases which produce unexpected thrombosis and meningitis due to softening, or osteophlebitis of aberrent vascular communications. If there is a well-defined line of cells running down between the external auditory canal and the sinus we may have sinus thrombosis, just as in the thoroughly pneumatized mastoid, but you may not have the same symptoms, proper discharge, tenderness, swelling.

The point I wish to accentuate is this: The series of cases which I presented with skiagraphic findings were the old, chronic, catarrhal otitis media non-suppurativa with acute exacerbation. The skiagraphs show, without exception, marked arrest of pneumatization. The peculiarity of the mucosa accompanying arrested pneumatization and on which the arrest of pneumatization depends, according to Wittmaack, renders it especially susceptible to low, chronic types of inflammation. The work of Wittmaack has been gradually built up on eleven years of research and during the four years since its publication in Germany there has not been a word said disproving it. In the recently published transactions of the Deutsche otol. Ges. there was not one word of contradiction of Wittmaack's work, although the German's are particularly critical of each other.

REVIEW OF OUR KNOWLEDGE OF THE BLOOD VESSELS SUPPLYING THE INTERNAL EAR.

By GEO. E. SHAMBAUGH, M. D., Chicago, Ill.

I have been asked to prepare a statement of our present knowledge concerning the blood supply of the labyrinth. The subject is largely one of purely scientific interest but is not without its practical bearing on clinical otology. My own interest in the blood vessels of the labyrinth was aroused in the first place while studying cases of the apoplectiform type of internal ear disease ordinarily designated as Meniere's disease and where the presumption formerly was that the disturbances were the result of hemorrhages into the labyrinth. An increasing knowledge of the labyrinth and especially of the symptomatology of sudden disturbances of the internal ear, especially those in connection with invasion of the labyrinth by infection, such as the extension from suppurative otitis media, has established on a firm basis the proposition first advanced by Meniere, that a hemorrhage into the labyrinth is capable of producing the typical Meniere-symptom-complex. This symptom-complex consists of the sudden onset of deafness associated with tinnitus and vertigo. It has become equally clear on the other hand that this symptom-complex is produced by any sudden involvement of the internal ear, such as might result from traumatism, or from an extension of infection from some neighboring part as well as the toxic action of infection on the eighth nerve or the action of certain drugs.

Before entering into a discussion of the details of the labyrinthine blood vessels, I desire to call attention to certain facts in connection with this blood supply which should be kept in mind when considering the possible clinical effects of alterations in this supply.

The first point of interest in this connection is that the entire labyrinth is supplied by a single vessel which enters

the internal ear by way of the meatus acusticus internus. It is evident therefore that a tumor developing in the internal meatus might disturb the function of the labyrinth not alone by exerting pressure on the eighth nerve but by interfering with the blood supply of the several end organs of the internal ear. An embolus lodging in this artery would result in a sudden suppression of function and give rise to the Meniere-symptom-complex in its most characteristic form. The deafness would be complete and permanent and the vertigo intense, though of course the latter would be of but temporary duration.

A second anatomical fact of interest because of its possible clinical bearing is that the several divisions of the labyrinthine artery going to supply different parts of the labyrinth are in the nature of end-arteries, that is there exists no anastomosis with other vessels except through the terminal capillaries. This is the type of arterial supply to the brain and certain other organs. This anatomical fact makes it quite clear that an embolus, such for example as the air-embolus in cassion disease, when lodging in a particular branch of the labyrinthine artery will produce an infarct in the region supplied by this particular artery and cause a suppression of function of the end organs restricted to the region which this vessel supplies. For example, a single arterial trunk called the anterior vestibular artery is the sole blood supply for the macula acustica of the utricle and the crista acustica of the horizontal and superior canals. An embolus lodging in this artery will produce disturbances of equilibrium and vertigo, but will not affect the function of the cochlea. Every carefully observing otologist with any extensive experience has encountered just such cases clinically, which apparently might be caused by such a lesion as the one just described, or possibly by some selective toxic action affecting the vestibular nerve but not the cochlear branch of the eighth.

The arterial supply of the cochlea consists of a large number of stems which, leaving the main vessel in the modiolus, radiate at regular intervals out along the lamina

spiralis ossea to supply the organ of Corti which rests on the membranous extension of the osseus plate, known as the membrana basilaris. Each one of these radiating stems are actually end arteries an obliteration of which will bring disturbance to but a restricted area of Corti's organ. From our knowledge of the physiology of the cochlea we would conclude that such a lesion will produce a defect more or less restricted in the midst of the tone scale. Here again is a clinical phenomenon with which otologists are quite familiar. I have had opportunity to study several cases where individuals apparently in normal health and with no history of any previous ear trouble developed a sudden involvement of one ear causing tinnitus aurium with a depression of the hearing function and where the functional examination demonstrated a lesion evidently in the labyrinth causing a defect restricted to the lower end of the tone scale. The location of the lesion in the cochlea itself seemed all the more certain because in most of these cases the symptom of diplacusis was also observed. Such a clinical complex might be accounted for on the assumption of an embolus in, or a hemorrhage from the artery supplying the apex of the cochlea.

Another point in connection with the blood supply of the labyrinth, which is of considerable interest in its bearing on clinical otology has to do with the question of anastomosis between the vessels supplying the endosteum of the labyrinth, this is branches from the labyrinthine artery and those found in the osseous wall surrounding the internal ear. It is apparent that where such anastomoses exist we would have a plausible explanation for extension of infection from the middle ear to the labyrinth without the formation of a bony fistula or of a rupture through one of the labyrinthine windows for the reason that blood vessels supplying the mucous membrane of the middle ear cavities also supply the underlying bone. Politzer and others have believed that such communications do exist between the tympanum and the inner ear. Some years ago I devoted a special research to this problem and was able to dem-

onstrate a great many places where the labyrinthine artery sent branches into the surrounding bone. This condition was found however only in the calf. In neither the pig nor the sheep, both of which I was studying extensively at that time was there any evidence that the artery for the labyrinth gives off branches to the surrounding bone. Since that time I have worked out the blood supply for the labyrinth of the dog as well as the human and in neither of these have I been able to demonstrate any vessels from the interior of the labyrinth which supplied the bony capsule. This fact if true, closes the door for any possible communication between the blood supply of the internal ear and that of the mucous membrane lining the middle ear.

Another fact in connection with the blood supply of the labyrinth deserves special attention because of its fundamental bearing on the problem of sound perception. This is the fact that the membrana basilaris is always a vascular structure. It is true that the so called zona pectinata, that is the part of the basilar membrane lying external to the tunnel of Corti has in some of the animals thus far studied been found free from blood vessels. I found vessels in the zona pectinata in the pig, the sheep and the calf but I did not observe these either in the dog or man. Nabeya found the zona pectinata vascular in the rabbit but not in the guinea pig. The important point is that in all the animals studied, including man, the part of the basilar membrane directly under Corti's organ, that is under the tunnel of Corti, always contains blood vessels. The reason why the vascularization of the basilar membrane is of special interest is because this is the structure fixed upon by Helmholtz and his followers as the vibrating mechanism which by responding according to the principle of physical resonance in its several parts to tones of varying pitch, brings about the stimulation of different areas of Corti's organ for each tone of the scale. It is of course essential that this vibrating mechanism should always respond the same to the same impulse, otherwise there could be no recognition of pitch. It requires no argument to prove

that a vascular structure such as the basilar membrane is incapable of reacting the same at all times to the same impulse. So that whatever our conclusions may be in speculating on the function of sound perception, one thing is quite definite, namely that the hypothesis that the basilar membrane is the vibrating mechanism is rendered untenable because of the fact that this membrane is a vascular structure.

I desire to call attention to but one more fact in connection with the blood supply of the labyrinth which has an important bearing on clinical otology. We have in the membranous labyrinth, a highly vascular structure suspended in a fluid which completely fills these osseous chambers. In the bony walls of the labyrinth there exist several openings suitable for relieving an increase in the intra-labyrinthine pressure. These openings are the oval and round windows, the aqueductus cochleae and the aqueductus vestibuli. Through the latter passes the ductus endolymphaticus, a tubular prolongation from the membranous labyrinth which on the posterior aspect of the petrous bone opens into a veritable expansion tank apparently designed especially for taking care of fluctuations in the intra-labyrinthine pressure. From this description of the relations of the internal ear, it is apparent that with each pulsation of the heart there must be produced in the labyrinth a to-and-fro movement of the endolymph. The bearing which this fact has to clinical otology is as follows. One of the most important functions of the vestibular mechanism is to supply a constant stream of tonus impulses to the skeletal muscles so necessary in preserving the normal equilibrium. It seems probable that this tonus function of the semicircular canals is a much more vital and important function than that of giving information regarding turning movements and position in space. That these tonus impulses emanate from the crista acustica is generally accepted but what produces the constant stimulation of the hair cells of the crista has never been satisfactorily explained. Ewald ventured the suggestion that

these cells were kept in a constant state of stimulation as the result of endolymph currents which he assumed were constantly present as the result of the constant replenishing of the endolymph from the stria vascularis and its escape through the ductus endolymphaticus. It is only necessary to point out that such a current could not exist for the simple reason that the ductus endolymphaticus ends in a closed sack and does not communicate with the cerebrospinal fluid as Ewald assumed. Barany accepts the conclusion that a constant stream of tonus impulses emanate from the hair cells of the cristae but offers no explanation for the continuous stimulation of these hair cells which this hypothesis presupposes. The explanation given above of a continuous to-and-fro movement in the endolymph resulting from the arterial pulsations supplies exactly the sort of stimulation necessary to account for the constant supply of tonus impulses which as we have already pointed out constitutes such an important part of the vestibular function. In my work on the structure and function of the crista ampullaris (1) I offered this explanation of the origin of labyrinth tonus and at the same time showed how the sum total of tonus impulses from the labyrinth on one side stimulates the muscles causing movements toward the opposite side thus accounting for the clinical phenomena observed in the sudden destruction of one labyrinth when the patient falls toward the affected side and a spontaneous nystagmus develops with the slow component towards the affected side. I repeat the explanation here as it seems to be a point not fully appreciated by many otologists.

The facts may be set forth briefly as follows: My studies of the minute anatomy of the crista ampullaris convinced me that an actual displacement of the cupola on the crista as generally supposed, where-by all the hair cells on each side of the crista are stimulated is not possible. This hypothesis of displacement of the cupola had given rise to the

(1) Uber den Bau und die Funktion de Crista Ampullaris. Zeitschrift fur Ohrenheilkunde, etc., Mai, 1912.

theory of a positive and negative response for the hair cells of the crista depending on the direction of the endolymph current. This is not sound physiological reasoning to assume that the same hair cells are capable of one kind of response by an endolymph current in one direction an opposite kind of response by a current in the opposite direction. With my conclusion that a displacement of the cupola is impossible it is apparent that only those hair cells on the side of the crista receiving the impact are stimulated by an endolymph current while the hair cells on the opposite side of this crista are stimulated by an endolymph current in the opposite direction. With this fact in mind the explanation why the sum total of the tonus impulses from the right labyrinth produce movements towards the left and vice versa becomes quite clear. The explanation is as follows: It has been established that an endolymph current in one direction in a semicircular canal stimulates the muscles which produce movements in the direction of the endolymph current while a current in the opposite direction stimulates the opposite group of muscles. Furthermore it has been established that for each canal an endolymph current in one direction produces a much stronger response than does a current in the opposite direction. It happens now that for each canal it is those endolymph currents which stimulate muscles resulting in movements towards the opposite side that give the stronger responses which is only another way of saying that the impulses emanating from those hair cells of each crista, the stimulation of which produces movements toward the opposite side, are stronger than the impulses from the opposite group of hair cells which produce movements towards the same side. The tonus impulses from the crista of each canal stimulates muscles causing movements in the two directions but the stronger impulses are of course always those causing movements towards the opposite side. Barany in a recent communication (1) reports a series of observations bearing on the effect of the

(1) Modern Labyrinthology—Laryngoscope, July, 1921.

vascular supply of the labyrinth on labyrinth tonus.

On page 409 he states that "this (phenomena of ocular movements) is due to the to-and-fro movements of the endolymph and that this movement is brought about by the conjestion or depletion of the vessels in the labyrinth. It may be assumed that the endolymph is directed towards the vent during systole and away from it during diastole. As the result of this observation I think the hypothesis of Shambaugh advanced some years ago may be realized, namely, that the pulse wave in the normal subject influences the endolymph in a to-and-fro motion and thereby establishes the tonus of the vestibular apparatus from the periphery. Apparently these movements are so slight, due to the small calibre of the normal vessels, that they do not produce a slow ocular movement in the normal subject, nevertheless I think it might be possible to maintain tonus of the vestibular apparatus from the periphery with even such a slight stimulation. In the presence of hyperemia of the labyrinth these movements naturally are more marked and then set up this rocking ocular movement. The presence of a peripheral labyrinth tonus (Shambaugh) does not indicate, as I would observe in passing, a coexistent central tonus of the nerve cells." With this brief statement of the bearing which the blood supply of the labyrinth has on clinical otology, we approach with added interest the study of these blood vessels.

The Arterial Supply.

This comes from a single vessel, the labyrinthine artery which reaches the internal ear by way of the meatus acusticus internus. I have seen no exception to this arrangement except in the calf, where as an anatomical variation an arterial trunk was seen to penetrate the capsule of the labyrinth and form the artery of the crus commune.

As regards the distribution of the branches of the labyrinthine artery to the several parts of the labyrinth, a striking uniformity exists for all the animals so far studied. In

each species including man the first branch constitutes the sole blood supply for the macula acustica of the utricle as well as the cristae acusticae and the anterior crurae of the horizontal and superior semicircular canals. This vessel is known as the anterior vestibular artery.

The crista acustica of the posterior canal is supplied by the second branch known as the posterior vestibular artery. This vessel supplies not alone the crista acustica of the posterior canal but also the crus commune, the posterior crurae of the posterior and horizontal canals and both crurae of the posterior canal. There are a number of variations in this arterial supply for the crus commune and posterior canal. The most typical arrangement is for a single artery to supply all of these structures. In some animals as for example the sheep and the calf, the posterior vestibular artery supplies only the crista and the posterior crus of the posterior semicircular canal, while a separate branch from the labyrinthine artery supplies the crus commune and the posterior crurae of the superior and horizontal canals as well as the anterior crus of the posterior canal. The position of the artery supplying the semicircular canals is always the same. They lie in the endosteum along the concave side of the canal.

The arrangement of the arterial branches at the base of the cochlea differ widely in different species. In both the calf and the pig an elaborate system of anastomosing loops exists from which are given off all the arteries to the cochlea as well as to the vestibule and semicircular canals, with a single exception the anterior vestibular artery, the first branch of the artery of the labyrinth is given off before the vessel breaks up into anastomotic loops. In the sheep these anastomotic loops are very meagre as they also are in the dog and in man.

The typical arrangement for the arterial supply of the cochlea consists of two sets of radiating arteries. One set runs out along the lamina spiralis to supply the spiral ganglion and the organ of Corti, the terminal twigs forming one or two so-called spiral vessels attached to the under surface

of the basilar membrane directly under the tunnel of Corti. From these spiral vessels an occasional branch radiates across the zona pectinata of the basilar membrane to join the vessels in the spiral ligament. These vessels in the zona pectinata I found in the pig the sheep and the calf but not in the dog or in man. Nabeya found these in the rabbit but not in the guinea pig.

The situation as regards the vascularization of Reissner's membrane is peculiar. This structure remains as a rule free from blood vessels. In some species however, it is a vascular structure. In the human labyrinth no vessels are found in Reissner's membrane. This is also true for the domestic pig, the guinea pig and the dog. In the sheep, the calf and the rabbit, Reissner's membrane is vascular. The arterial supply comes either from the vessels radiating over the scala vestibuli or from branches from the stria vascularis while the veins join those of the lamina spiralis.

The under surface of the basal coil is usually quite free from arterial vessels. In the sheep and the calf, I found a few arterial twigs lying in the endosteum of the basal coil, external to the veins occupying this region. In both of these species an arterial twig is constantly found leaving the labyrinth along the aqueductus cochlea. It is not unusual also to find a small arterial twig leaving the labyrinth along the aqueductus vestibuli. I found such a vessel in both the sheep and the calf, but did not observe this in the pig, the dog or in man.

The Venous System.

The venous system of the labyrinth can be summed up in a very few words. In most of the species so far studied all the veins from the labyrinth are collected into a single vessel which leaves the internal ear by way of the aqueductus cochlea. This condition exists in the pig and the sheep as well as in the guinea pig, the dog and the cat. A variation from this rule is found in the rabbit and in man, where the chief exit of the veins from the semicircular canals is along the aqueductus vestibuli. In the calf a

single vein, that of the aqueductus cochlea is the rule, and as an anatomical variation, occasionally a small vein from the crus commune leaves by way of the aqueductus vestibuli. Siebenman for the human ear and Asai for the dog and the rat have described a third vein which drains the labyrinth lying in the meatus acousticus internus. Neither Nabeya nor myself have been able to verify this vessel. No vessel leaving the labyrinth was found by me in the internal meatus of the pig, the sheep and the calf. I have since made a series of preparations for man and the dog. In both I found all the veins leaving the labyrinth along the aqueductus cochlea or the aqueductus vestibuli. Nabeya found in both the guinea pig and the cat that all the veins of the labyrinth were collected into a single trunk, the vein of the aqueductus cochlea. In the rabbit only did he find a vessel draining the semicircular canals which found its way through the aqueductus vestbuli.

A summary of the more important features of the blood supply for the internal ear includes the following points:

(1). The arterial supply is from a single vessel entering the labyrinth by way of the meatus internus. An exception in the nature of an anatomical variation was found by me in the calf where an artery from the bony capsule supplied a branch to the crus commune. A similar observation was made by Sappy.

(2). The typical arrangement for the venous system is for all the vessels to collect into a single trunk which leaves the labyrinth along the aqueductus cochlea. An exception to this arrangement is found in some species including man, where the veins from the semicircular canals in part or as a whole are collected into a trunk which leaves along the aqueductus vestibuli.

(3). No communications exist between the blood supply of the labyrinth and the surrounding structures. Exceptions to this rule occur occasionally. In the calf many branches from the labyrinthine artery were given off to the bony capsule. In the sheep, the calf, and the dog, a

small arterial twig left the labyrinth along the aqueductus cochlea.

(4). The arterial supply to the several parts of the labyrinth is through end-arteries. The several end-organs isolated in this way are: (a) the macula acustica of the vestibule, and the crista acustica of the superior and horizontal canals; (b) the crista acustica of the posterior canal; (c) the several parts of Corti's organ, each supplied by a vessel which radiates out from the modiolus along the lamina spiralis; (d) the macula acustica sacculae.

(5). The membrana basilaris is always a vascular structure, blood vessels being constantly found under the tunnel of Corti. The importance of this fact is that these vessels make it impossible for this structure to fill the role of the vibrating mechanism in sound perception, since to fill this role it is essential that it should vibrate the same at all times to the same impulse, and this a vascular structure would not be able to do.

Discussion.

Dr. J. Gordon Wilson, Chicago: The Society is indebted to Dr. Shambaugh for this demonstration of beautiful and instructive preparations of the vascular circulation of the internal ear. I can follow Dr. Shambaugh on most of the deductions he makes, but there is one based on an interpretation of the anatomical preparations to which I take objection, namely, relative to tonus. Dr. Shambaugh's hypothesis can be stated briefly as follows: Labyrinthine tonus owes its origin to stimulation of the hair cells of the crista ampullaris and this stimulation is the result of a to and fro movement of the endolymph resulting from the heart beat. Parenthetically it might here be remarked that the evidence for the maculae is at least as clear, if not clearer, than for the cristae. On this hypothesis Dr. Shambaugh would explain the constant, steady and almost indefatigable force which, originating in the labyrinth, acts on certain muscles of the body.

My objection to this hypothesis may be thus stated briefly:

(1) The principal vessels in the labyrinth are capillaries with a few arterioles, and one gets a false conception of their size from injected vessels. The vessels here shown are not only enormously enlarged but also relatively dilated by the injections. The capillaries are microscopic and the arterioles are little larger. In no capillaries, in any part of the body where they have been studied, has pulsation been seen. "In capillaries the rhythmic flow of the heart is converted into a continuous flow." Pulsation seen in capillaries and veins is pathological. There has been much dispute as to whether or not in arterioles of the size present in the labyrinth pulsation is present. Many believe it is not. In the eye whose vascular constitution closely duplicates the ear, even though the vessels are larger, pulsation is never seen except in pathological states. (Fuchs).

(2) There is no experimental evidence that such rhythmic movement as this hypothesis requires occurs in labyrinth fluids. Ewald did not observe any such pulsation after injecting a dye into the endolymph, though he noted a restless condition. He believed this to be a current in one direction and postulated movement by ciliary action.

(3) In pathological states involving the cardiac circulation one might expect to find some alteration in tonus in cases where one has so wide a gap between systolic and diastolic pressures as occurs in aortic regurgitation. Here the difference in pressure between systole and diastole may be as much as 9 times greater than normal, and yet no effect on tonus has been noted. What experimental evidence we have is not confirmatory of the hypothesis; in injection of drugs which greatly affects the blood pressure there is no alteration in tonus. In decerebrated rigidity we have a tremendous increase in tonus with little circulatory disturbance. To argue that such pulsation is so small that it cannot be seen or determined is so negative a

position that I am sure Dr. Shambaugh would be the last to suggest it.

On the other hand we have a mass of evidence pointing to gravity as being the casual factor. On this subject I have so recently written that I need not here refer to it.

Dr. Emil Amberg, Detroit: I think that otology is very much indebted to Dr. Shambaugh for this beautiful work. The idea that the basilar membrane is not instrumental in tone perception has been brought forward also by Oswald Baer in 1872, in a doctor's dissertation in Breslau. While I do not desire in any way to take issue with this idea, the mere fact that a blood vessel has its course in the lower portion of the basilar membrane, is to me no proof against the functioning of the membrane. It is by no means impossible that Dr. Shambaugh's investigations concerning the vessel supply, and the fact that the arteries are endarteries are of much greater clinical significance than the doctor is probably aware of. To my mind, the facts adduced go very well in hand, I may even say, they dovetail with Wittmaack's findings concerning otosclerosis. If that should prove to be the case, we see a corroboration of Wittmaack's views and we may stand at the threshold of new conceptions. While not exactly pertaining to the subject, may I be permitted, Mr. President, to touch on a few points which Dr. Shambaugh mentioned in his discourse? We learned that the doctor did not realize in the beginning of his work what it might lead to. Frequently in research work, new problems present themselves in the course of the work. Things which appear to be strictly theoretical may assume great practical importance in the future. This should be a great incentive, especially to the younger men. Furthermore, we learned that at a moment of rest a thought struck the doctor like lightening. That suggests to us that rest is essential for contemplation. Constant routine work does not allow thoughts to be developed, nor does it allow the development of the greatest

efficiency. The well know story of a gentleman who said he could do a year's work in 9 months, but not in 12, illustrates this point very well.

Dr. E. A. Crockett, Boston: Dr. Shambaugh made a very important clinical inference when he said that the apoplexy of endarterial type was the common cause of the apoplectic form of Meniere's disease. If you take a clinical report you will find very little diagnosis made, usually of the apoplectiform type. In a very large number of cases the Barany test will show both ears affected, one markedly, one slightly. These lesions are not in the labyrinth, but must be central. In a woman with severe apoplectiform vertigo, a careful Barany test showed inaccuracy of the labyrinth on both sides. Clinically speaking, the labyrinth is very seldom affected. I believe the lesion is extralabyrinthine and in the neighborhood of the pons.

Dr. E. P. Fowler, New York: Just one point, which I brought forward 12 years ago, as a possible solution of the tonus problem: namely that possibly convection currents of or in the endolymph might be the cause of the tonus. This was not taken seriously at the time but there are many facts which might support that theory: the constant presence of such currents as proved by physical laws and the preponderating influence of the vertical canal. We have better tonus with the head erect. In other positions there is a partial failing of tonus. I showed in a certain case how minute temperature changes would influence vertigo and nystagmus, (even with two to three degrees Fahrenheit), by means of caloric irrigations in certain selected cases.

Dr. Edward B. Dench, New York: As I understand it there is no communication between the vessels of the labyrinth itself and the paralabyrinthine tissue. Is there any explanation for the symptoms of perilabyrinthitis,

which immediately ceases when you remove the bone? Some vessels come very close to the perilabyrinthine bone and certain infections might change the chemical composition of the fluid or unduly affect these tissues. You do find abnormality of the bone which lies between the semicircular canal, and by cleaning that out symptoms disappear.

Dr. Isaac H. Jones, Los Angeles: The anatomic data Dr. Shambaugh has just given us helps clear up the diagnosis of a recent case. A woman, in excellent health, sitting quietly at an afternoon tea, was stricken with vertigo and vomiting, carried home and put to bed. The vertigo and vomiting continued for four days; then, as she expressed it, "I opened my eyes and saw double." Complete recovery —with exception of the following unusual finding; both cochleas normal; all responses from all semicircular canals normal, except left horizontal canal. Caloric stimulation of left horizontal canal produced no response whatever. On tilting head forward, nystagmus and vertigo appeared at once; on tilting head back again 60°, no responses. Dr. Ingham and I figured that the lesion is adjacent to the left VI nucleus in the pons, presumably embolus in a minute arteriole. Dr. Crockett just mentioned that he has come to consider such apoplectiform disturbances as usually of central origin; in this case it would seem improbable that both cochleas could be normal and all semicircular canals normal except one and that this one isolated attack had caused a destruction of the left horizontal canal itself. Again, the involvement of the left external rectus seemed to determine that the lesion is not within the internal ear. Reinforcing the concept of this lesion as being within the brain-stem, comes this demonstration by Dr. Shambaugh, that the superior and horizontal canals are supplied by the same artery. An embolus or other lesion affecting this artery would presumably not have destroyed function in the horizontal canal without affecting the function of the superior canal.

Incidentally, this case suggests that the pathway from the horizontal canal traverses the region near the VI Nucleus of the same side.

Dr. B. Alexander Randall, Philadelphia: In spite of Dr. Shambaugh's beautiful demonstration, I still hold to the views of Helmholtz. I recognize no other structure in the cochlear so capable as the basilar membrane of furnishing resonance to the Corti apparatus. This arterial theory is not sound. With a vibrating string you can touch nodal points without interfering with the vibration. The vessels may do no more than mark the nodes in the vibrations of the composing striae of the basilar membrane.

In the stria vascularis of the cochlea we have a secretion of endolymph. I do not believe that this is the source of that in the semicircular canals which acts upon the crista. The preponderant effect of the current is in one direction—in the superior canal, the upward being the most powerful, for upon the corresponding side of the crista we have twice as many hair-cells as on the other. In the convexity of each semicircular canal we have a corresponding organ which differs from the rest of the lining of the canal. At such points we have a distinct secretion of the endolymph, which would provoke a current in the canal towards the ampullae and give an increased hyperemia flow, which impresses itself upon the hair-cells of each crista. I believe that is of fundamental importance.

Dr. G. E. Shambaugh (closing): I have been interested in seeing how the anatomical facts impressed people. I wonder how we get these results if blood vessel communications do not exist. In answer to Dr. Crockett, hemorrhage can have that effect, but it is usually something else. I like to get Dr. Wilson's criticism, but I think he leans over backwards to find objections. I do not see why pulsation cannot exist in the labyrinth. I think there is increased pressure with the cardiac systole. Barany

thinks that with cardiac lesions there is a variation there. When one ear is destroyed, if you increase the tonus why is there any disturbance. After a long time both come equal, but in recent cases they are not equal.

In answer to Dr. Randall: It is perfectly possible to see pulsation in the eye grounds, sometimes in apparently normal cases, although I have never been able to get a sign of the pulse passing into the capillary system.

Replying to Dr. Dench: We would find a more ready explanation for the symptoms occuring in cases where we diagnose perilabyrinthitis were it established that blood vessel communications exist between the membranous labyrinth and the bony capsule.

Regarding the remarks by Dr. Crockett: Hemorrhages can readily account for internal ear disturbances but I am inclined to think that the cases where an actual bleeding in the labyrinth takes place is very exceptional. The apoplectic attacks of internal ear disturbance such as occurs, for example, causing the Meniere symptom complex are perhaps more often embolic processes.

I was pleased to hear Dr. Wilson's criticism especially, because it is apparent that he was making a strained effort to find some objection to the hypothesis which I advanced that the pulsations in the labyrinth could account for the origin of labyrinth tonus. I do not feel that he made a very good case. It is apparent from the descriptions I have given of the anatomical relations that such pulsations do take place. An actual demonstration can of course not be made but this in no way argues that they do not exist. Barany from his clinical observations seems to feel that the explanation which I offered years ago for the origin of labyrinth tonus is the most likely explanation of this phenomena.

Dr. Randall stated that it is perfectly possible to see pulsation in the eye grounds, sometimes in apparently normal cases, although he has never been able to get a sign of the pulse passing into the capillary system.

RECONSTRUCTION OF CONGENITAL DEFECTS AND ACQUIRED EXTERNAL EAR DEFORMITIES.* (ABSTRACT).

By JOSEPH C. BECK, M. D., Chicago, Ill.

In presenting the subject of Otoplasty as titled in this paper, the writer is compelled to resort to brevity. Therefore all technical details as well as references to literature will be limited to the minimum. Departure from the usual style is that the conclusions are presented first and again last.

A series of lantern slides of cases in various stages of operation (376 illustrations) from which one will get the idea of the difficulties entailed in the management of cases of otoplasty. The subject of correction is divided into those (a) Congenital origin and (b) that of acquired. These are outlined in the following table which the writer has prepared for a working basis:

A. Congenital
- I. Complete absence of one or both auricles.
- II. Absence of part ear.
- III. Asymmetry of the two ears.
- IV. Accessory or multiple ears (Poliotia).
- V. Abnormally large ears (microtia or otomegally).
  - 1. of the Cartilaginous portion.
  - 2. of the Lobule (length or width).
- VI. Abnormally small ears (microtia).
- VII. Abnormal form but normal size.
  - 1. Flattening of the periphery.
  - 2. Cleft lobule.
- VIII. Abnormal protrusion of auricle (Otopostasis).
- IX. Drooping ears (Roll or Dog Ears).
- X. False position of the auricle (Heterotopy).
- XI. Adhesions of the ear (Synechia).

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*From the clinic of Drs. Beck and Pollock, North Chicago Hospital.

B. Acquired
   I. Complete or partial absence due to:
      1. Traumatic or mechanical destruction.
      2. Thermal.
      3. Chemical.
      4. Disease—
         (a) Infectious.
         (b) Malignancy.
         (c) Symptomatic.
   II. Enlargements of Ear:
      1. Hematomata.
      2. Inflammations.
      3. Neoplasms.
   III. Abnormal Protrusions.
   IV. Abnormal form but normal size due to:
      1. Shriveling following perichondritis, infected hematoma or abscess.
   V. Adhesions of the Ear after abscess, etc. Cicatrization from burns. Postoperative adhesions.
   VI. Postauricular deficiencies—Retroauricular fistulae following radical mastoid operations.

Conclusions.

(1) That the subject is very little treated in most of the text books, especially those written by otologists. The number of periodicals that appear from time to time are also of comparatively little value to any one that is devoting a great deal of time to plastic surgery.

(2) It requires a great many steps before one obtains the best result.

(3) At its best a reconstructed ear is a very poor imitation of the normal ear.

(4) One of the most important conclusions the write has come to is that in congenital cases, there are other congenital defects usually present especially about the cardiovascular and renal system and therefore repeated operations are not well withstood.

(5) Congenial ear reconstructions performed early in life do not keep pace in the growth with its opposite fellow and many times the same side of the face is retarded in the growth (hemifacialatrophy).

(6) In traumatic cases it is much easier to obtain better results and the late war has aided a great deal along these lines.

(7) Artificial prothesis is far superior to any ear reconstructed by surgery so far as appearance is concerned but the patient as a rule refuses to wear it even though it may be held perfectly in place. This is in accord with nasal prothesis in deficiency of the nose.

PRESENTATION OF A NEW INSTRUMENT FOR DETERMINING THE AMOUNT AND CHARACTER OF AUDITORY SENSATION.

By EDMUND PRINCE FOWLER, M. D., New York City, and (by invitation), MR. R. L. WEGEL, Research Laboratory of the American Telephone & Telegraph Co., and the Western Electric Co.

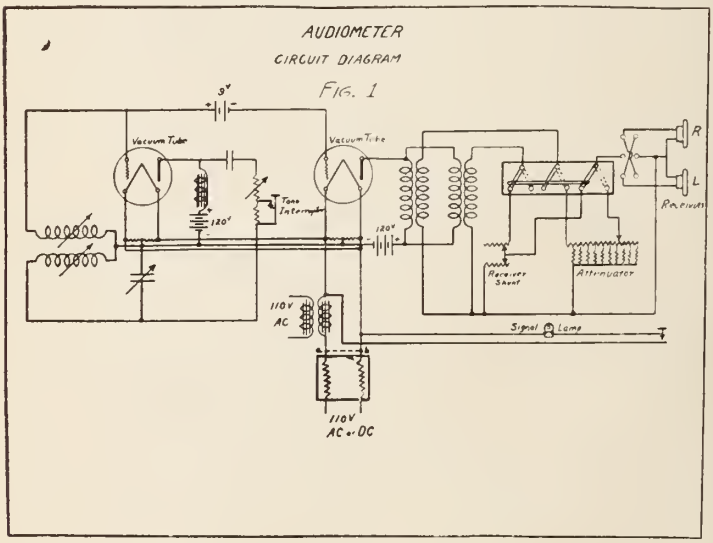
This work in collaboration with the physicists of the Western Electric Co. in the research laboratory was done with the idea of building a machine, not only of practical use to the otologist, but for investigating scientifically the acoustics of the human ear. The machine produces a pure tone range throughout a frequency scale from 64 to 16,000 cycles. The intensity scale ranges from below audibility up to where the sound is painful.

Mr. Wegel will describe the machine and briefly explain the foundation upon which we have built our charting.

"The audiometer which we have used in our tests consists, like most other modern audiometers, of a source of alternating current of variable frequency, an attenuator and a telephone receiver. The diagram marked Fig 1 is a circuit diagram. The electric source consists of a vacuum tube oscillator constructed with particular care to produce as pure a frequency as can be had with such apparatus. From the standpoint of purity of tone it is perhaps most important that oscillating coils and condensers of extremely high time constants be used. This precludes the advisability of attempting to use coils or condensers which can be varied gradually, since this always introduces impurities. The circuit is arranged so that the filaments of the tubes can be lighted by either alternating or direct current. A signal lamp is attached in such a way that the patient may signal the operator when he hears a tone under the control of the operator by means of the tone interrupter. This method of obtaining points was found to be most satisfactory because it requires a minimum of effort on the part

of the person being tested. The attenuator consists of a series of resistances arranged in the form of a special potentiometer in such a way that the current in the receivers may be varied in the ratio of about one to ten million. The resistances marked "receiver shunt" are provided so that tones may be produced which are louder than those obtainable on the attenuator.

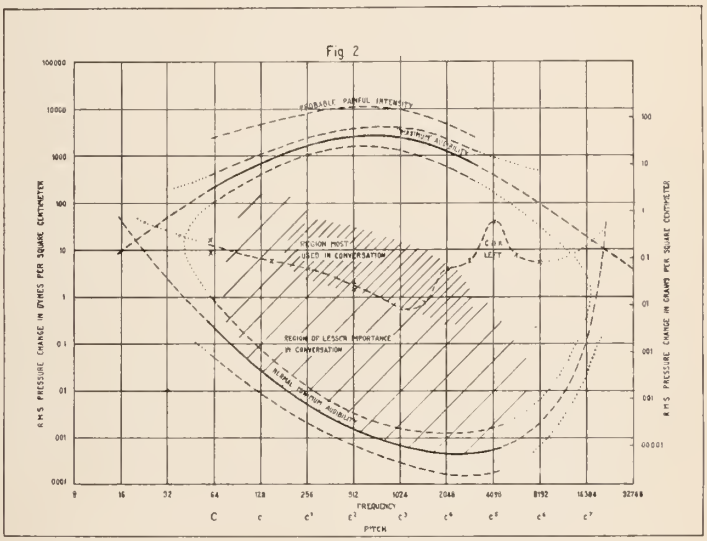
In presenting the audiometer it is hardly reasonable to omit a discussion of the philosophy of audiometry upon



which the design and construction methods are based. Considerable uncertainty is involved in the interpretation of functional tests of hearing. It is, therefore, advisable to look into the share of responsibility for this uncertainty chargeable to the methods and technique of measurement and to the kind of logic used in the interpretation of such tests. In order to make clear our conception of the problem it will be best to discuss briefly a few diagrams.

The diagram labeled Fig. 2 may be considered as a plot of intensity of stimulus at various frequencies. The

scale of frequencies corresponds to a uniform logarithmic scale exactly the same as with a piano keyboard. The stimulus is labeled R. M. S. pressure change in dynes per square centimeter. This means the effective pulsating pressure impressed on the ear-drum by the sound wave at the various frequencies. The curve marked "normal minimum audibility" indicates at the various frequencies the pulsating pressure in the sound wave required to produce the minimum audible sensation for the average of a large



number of ears. Part of this curve drawn with a heavy line represents data given in a paper before the last Chicago meeting of the National Academy of Sciences. The upper frequency part of this minimum audibility curve is drawn so as to be in agreement with data taken by Mr. C. E. Lane at the University of Iowa. This curve shows that the ear is most sensitive to the pulsating pressures of sound in the region of perhaps one thousand to five or six thousand cyles, or double vibrations per second. The dotted lines running more or less parallel to this minimum audibility

curve represent the so called "standard" deviations from the mean. The determination of the normal curve referred to and variations of normal ears will be taken up in as much detail as the data here represented warrant in a paper to be presented before the Triological Society this week.

If the sound pressure on the ear-drum is increased gradually a point may be reached at which a sensation of feeling very much like a prickling sensation will be produced. Sounds slightly louder than this are very painful. The curve marked "maximum" audibility in Figure 2 represents the pulsating pressure required to produce a sensation of feeling. It may perhaps be more logically called the "threshold of feeling curve." While it does not represent maximum audibility in the sense that it is the loudest sound that can be heard, it may be defined arbitrarily as maximum audibility because it represents a sound intensity which cannot practically be exceeded without producing a sensation of pain. It is impossible to say whether it corresponds to a stimulation of some of the nerves of the middle ear or to an over stimulation of the auditory nerve. The dotted lines running parallel to this curve show the "standard" deviation from the mean. Since these lines are closer to the "maximum" audibility than the deviation lines are from the "minimum" audibility it may be seen that the former is more definite than the latter.

There is considerable confusion concerning the lower and upper pitch limits of audibility. In regard to the lower point, for example, it is doubtful sometimes whether the vibrations are heard or felt. It may be seen that this diagram affords a definite though arbitary method of determining the average lower frequency limit. The minimum and maximum audibility curves are extrapolated to points of intersection. This point represents a frequency at which a sound wave can, on the average, be felt as easily as it can be heard. The sensation of feeling at the lower frequencies is milder and much less definite than it is at the higher frequencies. A similar point of intersection is

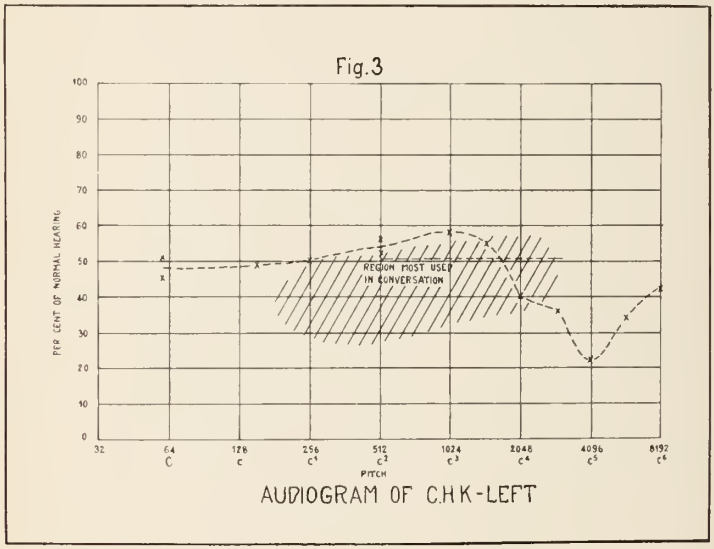
shown at the upper frequency ends of these curves. Looking at the figure as a whole, then, we may regard the ear as having a sensation of feeling which is more acute than that of hearing at all frequencies below the low point of intersection and at all frequencies above the high point of intersection, and that the sense of hearing is more acute between these points of intersection. The actual frequencies of these points have not been determined with enough certainty to definitely give the limits so defined.

The representative minimum audibility curve marked "C. H. K. Left" is given to illustrate the relation of a moderate deafness relative to the other curves in this diagram. The case was diagnosed as a fairly well advanced catarrhal deafness. The man was capable of carrying on a conversation without artificial aid if the speaker was comparatively close to him and talked either in a normal tone of voice or slightly raised voice.

The shaded portions of Figure 2 show the stimuli corresponding to the frequency mixtures as ordinarily used in a conversation, since speech may be regarded as consisting of a complex mixture of frequencies stimulating the ear simultaneously and varying in intensity. The difficulty encountered by C. H. K. in interpreting speech is made clear by the fact that an appreciable portion of the stimulus ordinarily used in conversation is inaudible to him.

According to the best information the nearest approximation to the relation between stimulus and sensation is logarithmic (Weber's Law and Fechner's Law). The scale of stimulus, that is pressure, in Figure 2 is plotted logarithmically so that equal divisions on the chart correspond to equal changes in sensation. The area included between the normal minimum and maximum audibility lines may be regarded as the normal area of sensation. Since no stimulus falling below the minimum audibility current for "C. H. K. Left" produces a sensation, his area is considerably smaller than normal if we may assume that his threshold of feeling is nearly normal. Since his curve passes nearly through the middle of the normal area the statement may

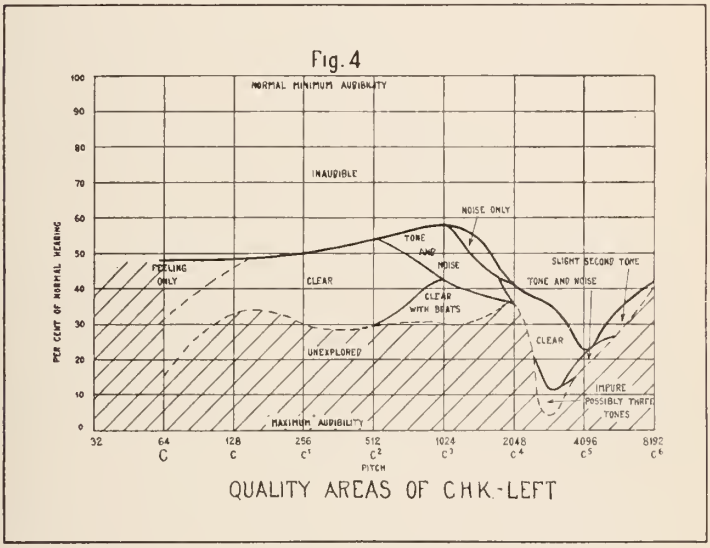
logically be made that he has lost one-half of his auditory sense. This forms one logical basis for defining "percent loss of hearing." The ordinary method of defining this by means of the ratios of distances at which a watch tick or whisper can be heard is in no sense logical and can only be justified by the fact that up to the present time there has been no other method. If the distance between the minimum audibility and maximum audibility curves at any frequency be taken as a measure of the capacity of the



normal ear to sense sound and the distance between the abnormal minimum audibility curve "C. H. K. Left" and the maximum audibility curve be taken as the capacity of C. H. K. to sense sound, then the ratio of the two may be defined as the "percent of normal hearing" retained by C. H. K. This is plotted in the diagram of Fig. 3.

This method of plotting audiograms and the foregoing discussion show the logic by means of which the conclusion has been arrived at that "percent of normal hearing" is one, at least, of the most logical scales for audiograms.

Among the other many characteristics of normal audition which are impaired by disease or injury is the quality of sensation. Some people are able to hear sound quite well but still are unable to distinguish what is said. It is entirely possible that measurements of the quality of hearing will be of as great significance as measurements of the quantity. The normal ear hears tones from a properly constructed audiometer which may be described as pure, simple, clear, even, musical, etc. Some abnormal ears do



not have this normal sensation but describe pure tones as producing vibrations, rattles, hums, roars, etc. These symptoms appear to be associated with so called catarrhal conditions which are active at the time of testing. The areas in Fig. 4 show the parts of the range for which the sensation of C. H. K. was abnormal."

Discussion.

Dr. C. E. Seashore, National Research Council, Washington, D. C., (by invitation): Otologists are to be congratu-

lated on the taking up of an entirely new point of approach to their science by the introduction of measuring instruments of precision. Not only can those problems be solved which we now have in sight, but unforeseen and unexpected ones are likely to come in the employment of instruments of precision. Having employed such instruments in the acoustic laboratory for many years, I am confident in predicting splendid returns to otology from this new approach not only from the scientific point of view, but also in diagnosis and treatment. I think it is a hopeful sign that the practitioners are getting together on these questions and joining hands with men of science.

In the practical work, we need the point of view which can be obtained only in more or less exact, pure science. For example, in evaluating the voice test for diagnostic purposes, the problem must be approached from the point of view of physics, psychology, and otology. Indeed, otologists of the future will need a more particular training in the psycho-physics of otology than heretofore. We cannot expect to study the functions of the ear until we can record with precision the amplitude, frequency, duration, and form of the sound waves; and we cannot make these measurements on human beings without having training in the recognition of the very numerous subjective and objective factors which determine hearing.

Let me mention a few of these: Attention moves by peaks; therefore the matter of preparation is important. If you give a sound outside of the peak of attention, the person tested is at a decided disadvantage. We must, therefore, control expectant attention. Another phase of this subject is that of hallucination. It is commonplace to demonstrate in psychological laboratories that people may be led to firmly believe and assert that they actually hear what they merely expect to hear under certain conditions. We have demonstrated this on men most distinguished for their powers of reliable observation. Fatigue is another factor. Efficiency of listening goes by waves—long waves and short waves within these—just as

in color vision there is a constant fluctuation in the color sensitivity of a given portion of the retina. Wave phase presents one of the most fascinating factors that we must control because, by determining the phase of the sound wave which shall strike the ear, we can make the sound appear to move in any direction in a predictable way and can split it up into its components, making each harmonic move in its own path—all illusory and yet in accordance with rigid law.

Mr. Wegel's chart is a beautiful piece of work; but such procedure is very treacherous. There is a fascination to a straight line and a satisfaction in a simple conception. But, if the simplicity of the conception covers up a number of uncontrolled factors, it is dangerous. In our own work, we have preferred to plot raw curves as norms without attempting to introduce the corrections as Wegel has done.

I would like to ask whether it would not have been better for Mr. Wegel to find out how loud a tone must be before it ceases to increase in loudness instead of setting the limit which he does. The upper limit must be where we cannot psycho-physically make the sound any stronger.

One word in regard to gaps: If the gap is narrow, it tends to fill up. When the sound is loud, the neighboring elements in the ear will be affected. It is therefore probable that physical gaps are much more numerous than our measurements would indicate

There are now two types of instruments in the field. One of them was originated in the University of Iowa and we have spent much money and energy on the problem. The other method is that illustrated by Dr. Wegel's measurements. We are using that in Iowa for the determination of the upper limit of hearing. There is, however, a fundamental difference in the measuring technique in the use of the two instruments. In our pitch range audiometer, we can sweep from the lowest to the highest audible tone by a gradual turning of a lever in a single sweep. Repeating this for the several intensity steps helps to shorten the test, saving the time of the otologist, avoiding fatigue of

the patient, and making a more complete survey than is possible by the other type of instrument in which measurements are made only at selected pitches.

I hope that the time may soon be at hand when we may have an instrument so simple that it can be put into every school to test the ears as eye tests are now made. In this way, incipient cases of disturbance could be detected at a time when they are amenable to treatment. The significance of measurements of this kind for civil service, army and navy, and various industrial purposes is apparent.

Dr. E. P. Fowler: As always the authors of the two papers to which we have just listened, have set before us information of absorbing interest. It is a great privilege to have the opportunity to discuss, though briefly, some aspects of the subject which, in view of late developments in the physics of sound, are destined to be of prime importance to the otologist.

In regard to Dr. Wilson's paper: I heartily coincide with all the views as summarized.

Using the instrument just presented before this society by Mr. Wegel and myself, I have obtained results differing somewhat from those reported by Dr. Wilson. (1) Whereas some unusual ears show well-defined peaks, such have not been chiefly near 1000 cycles, but distributed fairly evenly throughout the scale of frequencies. Moreover when more than one point of maximal sensitivity is prominent such may or may not be markedly beneath the greatest peak, about an equal number of times. (2) In order to learn why certain ears vary in their points of maximal acuity, it will be necessary to carefully correlate the audiometer findings with careful observations as to the anatomical and physcial conditions in each ear tested. It is probable that many variations can be proved to be due to differences in the vibrating characteristics of the transmitting mechanism of the ear—especially as regards: (a) area, thickness, tension, mobility, retraction and shape of the tympanic membrane: (b) size, weight, structure, position and sus-

pension of the ossicles: (c) condition of the ossicular articulations: (d) vascular changes in the middle ear (and cochlea): (e) size and resonance of the ear spaces and structures.

The method of plotting the audiograms is of paramount importance in this connection, as will be shown in a paper to be read before the Trilogical Society on May 4th.

Tinnitus in obstructive and nerve deafness is a fascinating field for study, and we too are beginning to glimpse certain very suggestive relationships by the use of accurate audiometric measurements. I believe with Dr. Wilson that a close co-operation between physicists and otologists will surely produce real results in the discovery of, and therefore the alleviation of many defects of hearing.

In regard to the paper of Dr. Dean: It is impossible to estimate the minimum audibility from the audiograms shown by Dr. Dean, as the curves are not based on a definite unit of intensity. Each point plotted is relative to nothing in particular.

His method of plotting audiograms uses no definite frequency or intensity scale, and it would appear therefore that no usable correlation could be expected between the audiometric and otologic findings.

The occurrence of "absolute tone gaps" in his graphs indicates that these may be due to instrument peculiarities, or at least partly due to these, because with the audiometer described this afternoon I have found no absolute tone gaps in the several hundred cases examined. In fact, one of the outstanding characteristics of our curves is the lack of so-called extensive gaps, unless we include in this nomenclature, the depressions of the highest tones in certain lesions. The usual ear tests are unsatisfactory because one cannot with any approach to accuracy, know the amount of sound intensity reaching the ear. Take for example the Galton whistle.

In the first place many Galtons cannot be made to emit over 14,000 double vibrations and even with the more perfect whistles the higher frequencies are possible only if we employ from 20 to 30 pounds air pressure.

Slight movements near the ear may accentuate or annul its audibility owing to wave interferences in and about the ear. One, and sometimes, two other tones accompany the true tone of the instrument, and the patient may be powerless to differentiate between them. Nevertheless, if he does not hear the whistle, he is supposed to have nerve deafness. If you will close your ears very tightly it can be demonstrated in a moment that mere obstruction of the external auditory meatus can cut off the sound from a Galton whistle, especially if it is blown a little distance from the ears. Well, have you nerve deafness? No! for the moment you have obstructive deafness, and so may some others who do not hear it. The trouble is we have been obsessed with the idea that a lessening of acuity of hearing for the higher notes means nerve deafness. I do not believe this is necessarily true, and the graphs I have made substantiate this belief. However, for the present it is well to preserve an open mind as to the meaning of an audiogram. As for bone conduction, the only way to determine the truth as to its meaning, is to carefully correlate the bone conduction findings with data on histology, anatomy, otoscopy, tests, progress, pathology, etc. It is probable it will be of great assistence in diagnosis, but it too must be employed with definite units of intensity and frequency. This has not at present been done.

Dr. Edward B. Dench, New York: We flatter ourselves that we know something of the physics of sound, but after Dr. Seashore sat down I realized we know nothing. There are a vast number of errors that can enter in and it should be impressed upon our minds the great care necessary in our methods. We are repaid for coming to listen to Dr. Seashore's remarks. One point to be demonstrated is the fact of anticipation on the part of the patient. In the clinic the patient hears the big fork vibrate, because he sees it vibrate, and hears the whistle because he sees it. Anticipation plays a part in the sound heard. The patient should shut his eyes and then one should ask him to

describe the sound he hears. Sometimes the patient hears the same thing if you strike the instrument and if you stop it. If the patient cannot describe what he hears, he does not hear. One must also be careful that the sense of feeling is not mistaken for hearing. I would like to ask Dr. Dean why the upper limit is more accurate by bone conduction than by air. The ear is supposed to receive the sound better by air than by bone, unless there is disturbance of the sound transmission.

Dr. Emil Amberg, Detroit: A unit for a certain kind of sound has been mentioned by Wundt. A cork ball, weighing 1 milligram, falling from a height of 1 millimeter on a glass plate is heard in a distance of 91 millimeters. In January, 1901, I published in the Journal of the American Medical Association a "Normal Acoumeter" (demonstration of the apparatus). It is an instrument of precision. A steel ball weighing 1 gram falls from a certain distance and then produces a sound. The Hoover Steel Ball Corporation of Ann Arbor, kindly furnished me with steel balls weighing 1.0018 grams. This morning the Committee on Standardization of Hearing Tests reported. It would be regrettable if matters become more complicated. There is one point which may be interesting from a speculative standpoint. The question has been asked as to what degree the increase in intensity of noises could be perceived. Wundt mentions that the degree of difference in perception for light is 1/100; for muscle sensation 1/17; and for pressure and hearing 1/3. Does not the fact that the number for pressure and hearing is the same possibly suggests that, in the last analysis, touch and hearing are closely related? To speculate further, for a moment, is it not possible that the mooted question of the specific function of the basilar membrane and the assumption that the ciliated cells may be most instrumental in tone perception, may be approached in this manner? The comparative study of the development of the organ of hearing from the lowest animals upward, may be of great help. The consideration of fatigue is very

important. Politzer mentions the changes during the day. Another point is that older people do not hear high tones as well as young people. The co-operation of the psychologist, phsiologist and other scientists with otologists will no doubt bear good fruit.

Dr. Isaac H. Jones, Los Angeles, California: One appeal that is made by this demonstration is that such instruments of precision, making possible the charting of auditory tests, may lead us to detect auditory impairment of central origin. It is surprising to consider how little we know about the intracranial pathways of the auditory apparatus. Neurologists, otologists and other investigators are continually seeing all types of lesions of brain-stem and cerebrum; yet lesions which one would expect to produce an impairment of hearing, seem to produce little if any impairment. Auditory tests are so seldom of any value in intracranial localization. At present we seem to be dealing almost exclusively in terms of the end-organ.

Dr. B. Alex. Randall, Philadelephia: In regard to several points which have been brought up at this time, I will call attention to the investigation of Dr. Blake of Boston, who showed that the high limit is very often a question of the thickness of the tympanic membrane; with an opening experimentally made through the drum head we can hear much higher tones. A child can as a rule hear higher tones than older people.

Fatigue is a very important factor, not only in relation to industrial fatigue but also in clinical cases where tests would induce us to put patients upon specific medication because of loss of the high tones. There is an element of lessening and supplying the gaps by mental processes, which is lacking at times of special fatigue and may need to be discounted in our comparisons.

Dr. Max Goldstein, St. Louis: I plead guilty to being a mere otologist. The more I see of the audiometer, the

more respect I have for the tuning fork and Galton whistle. The audiometer is an electric and largely foreign way to approach the acoustic mechanism, which in the human is adapted to the voice and its composite tones. The audiometer curve looks like a temperature curve. The results cannot be obtained without the use of other accessory functional tests.

Dr. Dean in his splendid deductions has demonstrated that for differentiation he uses bone conduction and the tuning fork, and checks up his audiometer findings by the monochord and Galton whistle.

Mr. Wegel makes the obscure differentiation between hearing and the sense of touch. After all, hearing and sight are merely modifications of the sense of touch. Is it not possible in his normal scale that he is mixing up audition and tactile impression? Is that scientifically accurate for the otologist?

While Dr. Dench and his committee are working on the standardization of tuning forks, we have submitted three types of electric audiometers, three varieties of scales and curves and no approach toward standardization.

Dr. George B. McAuliffe, New York: Hearing should not be tested by contact. There should be an air gap through which the sound should be carried to test totality of hearing conduction and perception of sound. A person with catarrhal deafness can sometimes hear the telephone very well and yet he may be more deaf than one with slight nerve impairment.

Mr. R. L. Wegel (by invitation): I might say in passing that the feeling sensation corresponds to the pressure of the sound wave shown in the figure. As to mixing it with the sense of audition, it has no relation except that it serves as a convenient point to tie to—it shows the upper intensity limit. Professor Seashore said that he thought a more logical place to stop was one which did not become louder as the stimulus was increased. I do not think one

can get people to submit to that test. Recent experiments made at the University of Chicago show that Weber's law holds very closely throughout the entire range of intensity. The least perceptible increase in the pressure of sound is 5 per cent approximately, regardless of what the level is. As far as any physical data are concerned, there is no indication of such a point.

Regarding the question of pitch variation being better than the variation of intensity, we have a slide which I had planned to discuss but did not have time. The apparatus of Drs. Dean and Seashore determines a point by varying simultaneously both the frequency and intensity; Dr. Fowler's maintains a definite frequency and varies the intensity. It may be that the variation of both frequency and intensity is the most accurate if done in the proper way. It seems to me that the curves should be considered as transitions from one level of sensitiveness to another. The parts of the curves which are most important are most accurately determined by variations of intensity. Still, these questions remain to be settled. When you vary frequency, you must have very carefully graded apparatus.

In regard to anticipation of the sound: we have made measurements by having the operator control the tone. The patient has a push button with which he signals when he hears the tone. The operator tries to fool the patient. You can find a place where he always hears the sound and a place where he never hears it, and there is an intermediate area due to fatigue or lapse of attention. One must not be misled by confusing a change of sensation with a change of stimulus. If you take speech and reduce it to minimal audibility a man will not understand anything you say. If you increase the intensity 100 times speech is very intelligible, but if you increase to 101 times, the difference is hardly noticeable.

Dr. Seashore says there is danger in using the straight line to represent the hearing of a normal ear. It is merely a matter of plotting the data, whichever is simplest.

Dr. Dean shows audiograms with normal curves and subnormal curves. We think you can exaggerate the lower part of the scale and make something small look very large. We think it is necessary to adopt a uniform scale. It is more a question of sense than of stimulus.

Dr. George E. Shambaugh, Chicago: An interesting sidelight is gained on the existence of tone gaps by experimental work. A guinea pig, for example, exposed to a whistle of a certain pitch for several hours a day over a period of weeks, when examined histologically will show a definite circumscribed degeneration of Corti's organ. The area will vary with the pitch of the whistle used. An interesting feature is that the area undergoing degeneration is always a rather extensive one, so one must presume that the part of Corti's organ stimulated by a particular tone in the scale is overlapped extensively by the area stimulated by a different tone but one near the same pitch.

Dr. Dean in his discussion used the expression "duration of bone conduction so many seconds." This expression does not mean anything to me. One fork which dies out very slowly with this duration of bone conduction would indicate very much shortened bone conduction, whereas another fork which dies out rapidly in the same number of seconds would indicate a prolonged bone conduction. One should be very cautious about attempting to interpret functional tests as though they necessarily had anything to do with the alterations which one sees in the drum membrane. To show a chart illustrating the tone range in a case where there is a relaxation or so-called stiffening of the drum membrane and chain of ossicles is of course misleading. The alterations on the drum membrane which one sees, has so frequently nothing to do with the defects in the hearing, these being caused by deeper seated trouble, as for example, alterations about the oval window or changes in the labyrinth itself. It is especially common for one to observe a strongly retracted drum membrane. For

example, a condition left over from an exhausted tubal catarrh, but where there is no defect whatever in the hearing. I rely rather upon the results of functional tests, quite independent of the findings on the drum membrane, for making the diagnosis as to the location of the pathological changes which are causing the defect in hearing.

These tests of the tone range are particularly interesting because of the sidelight which they are able to throw on questions in the physiology of hearing. In solving the problems in practical diagnosis, in differentiating the cases where there is the possibility of some improvement by treatment from those where treatment can be of no assistance. I have not found anything in these tests, as yet, that seems to be of any assistance. Probably something may eventually be worked out, but so far they do not give any assistance in the problems of the practicing otologist. As for employing these tests in cases of progressive deafness, for example, in cases of otosclerosis, to determine from period to period what progress has been going on, it would seem to me that as far as any practical usefulness is concerned it would be just as logical to ask a man whose hair is turning gray to have some colored photography made of these changes, to be checked up from time to time. The checking up of the hearing that is of practical assistance can readily be made in a few seconds time.

Dr. Seashore: In regard to gaps, in the region of 80 to 92 vibrations in my left ear I have a gap for pitch discrimination. I can hear the tones perfectly, but I cannot discriminate. My threshold for discrimination is 8/10 of a vibration, but within the region stated I cannot tell any difference in pitch at all. I have elaborate evidence which leads me to think that this is rather common. In scientific otology it would be interesting to distinguish that kind of gap from the gap where they cannot hear tone at all.

Dr. J. G. Wilson (closing): There has been fully tested and answered the argument that the telephone receiver

applied to the ear introduces the factor of bone conduction. With the telephone receiver as used no bone conduction is present. After using the audion oscillator for over 3 years I am optimistic in regard to the outlook of some such method for testing hearing, for more accurate diagnosis and for estimating progress. I do not think the time has come to base general deductions on this work, especially in intracranial cases.

Dr. L. W. Dean (closing): The longer a tuning fork will vibrate with a given excitation the longer would be the bone conduction. We use the standard Bezold fork in all our cases and consequently the results secured in our cases may be compared. Any other tuning fork unless it had the same length of vibration as the one we use would give different results.

In using the audiometer we do not measure the exact hearing power for any notes. We do, however, get the approximate hearing power for all the notes within the range of the machine. When the curve shows a tone gap it simply means that with the instrument we use, the tones in the gap are not heard. If a similar instrument producing more intense notes was made, it is possible that more of these tone gaps would disappear and would appear only as disturbances in the curve made with the new machine.

I prefer to determine the upper limit by bone conduction because impacted wax in the external auditory canal, borac acid powder in the external auditory canal and pure middle ear lesions may all change the upper limit as determined by air conduction. The upper limit as determined by air conduction is not decreased more than 6000 either by blocking the auditory canal or by pure middle ear lesions. In the cases of chronic ottorrhea tested we found at least in one case that when the upper limit as determined by air was decreased to 6000 d. v. by bone, it was 14,000 d. v. This difference in our judgement was due to debris in the canal and tympanum.

THE ESTIMATION OF THE MINIMUM AUDIBILITY OF TONES.

By J. GORDON WILSON, M. D., Chicago, Ill.

The observations made within recent years by physicists on the minimum audibility of tones have awakened an increasing amount of interest among otologists who have been quick to recognize the important bearing such knowledge will have on their specialty. In introducing this subject it will be advantageous in a few words to define the scope of the problem as it presents itself in this discussion.

The Problem

It is scarcely necessary to say that this discussion is not concerned with the psycho-physical problem of how distinctive qualities of tones are aroused. As we are dealing so far as possible with pure tones, the resultant vibrations from several tones such as are transmitted from an orchestra as one complex (seen for example in phonographic records) and their analyses in the internal ear, do not concern us. Again, inasmuch as the human voice and speech though dependent on a pronounced fundamental are efficiently modified by distinct overtones present, the minimum audibility of the voice will not be referred to. The topic before us represents the basic problem and it is of far reaching importance in otology. On it later the other problems may be advantageously based. We recognize that the quantity of the impulse over a nerve may be increased or diminished; we are here dealing with the minimum quantity required to awaken recognition of a pure tone—that is the threshold of hearing. This threshold may be lowered by education, as is touch in the blind; but I am not concerned with this question but with the average minimum perception in the average individual.

It has long been recognized by physicists that the estimation of the minimum audibility of tones (the threshold of hearing) at all pitches is of prime importance to an understanding of the physics of audition. Within recent years the physicist has encountered many practical problems to which he has sought to apply such knowledge. The late war in this realm as in other realms of physics stimulated the energy of the pure scientist to practical problems in acoustics. We all recall the immense amount of work done to perfect apparatus to detect the presence of and locate the position of submarines. Again, the telephone service, now become one of the necessities of modern life, needs for the engineering of an adequate service a knowledge in absolute terms of the sensitiveness of the hearing of the average telephone user, and this has required in the telephone service a study of the threshold of tone perception.

Not only is this estimation important for the physicist but in otology the testing of the absolute sensitivity of the ear at various pitches is of importance: (1) To determine the degree of impairment of hearing; (2) to localize the pathologic changes, in which it has proved superior to our ordinary methods of testing, and (3) to estimate the results of our treatment.

The difficulties encountered in testing hearing are considerable, much greater than in testing sight. In testing sight we can use one standard, white light. In testing hearing we have no uniform sound to use as a standard. While it is true that both light and sound are vibrations, the eye is not able and is not called on to resolve light into its composite colors, and even if it were the range of such vibrations is small compared with the ear. In the ear such differentiation of vibrations is an inherent part of its function as an organ of sense.

For an accurate knowledge of the degree of hearing a patient possesses we must have an estimate of the range of hearing (quality of hearing) and the quantitative degree

at each pitch. It is known to all of us that hearing for one part of the scale may be seriously impaired while that of another part is hardly affected. Accurate results therefore can only be obtained by estimating quantitatively the hearing for simple tones at all pitches. When one remembers that the qualitative range may be estimated at from 20 cycles to 20,000 cycles, or higher, one recognizes the wide area to be covered. The difficulty of obtaining a suitable apparatus capable of examining the threshold of hearing over so great a range has proved the great barrier to this investigation. This barrier has now to a large extent been surmounted and we are entering on a new era of advance and only a rash man will predict what the final outcome will be.

In such an investigation there are three main questions or problems which present themselves and which may thus be outlined briefly:

(1) What is the minimum amount of energy required to produce the perception of pure tones at various pitches—the physical problem.

(2) What light do such determinations throw on the ear as an organ of hearing—the physiological problem.

(3) Given a mean audible threshold what variations take place in defects of hearing, this involving the further question at which pitches such variations occur,—otological problem.

In considering these questions it is obvious that the more perfect the instrument from the standpoint of the physicist the more accurate the knowledge obtained in regard to the minimum audibility. To obtain the threshold value in the normal ear it is obvious that a prerequisite is that the instrument used be capable of reducing the pitch over the scale beneath normal audibility. There are many factors which will vitiate the findings, e. g., resonances in the telephone receiver, the presence of extraneous noises tending to confuse the observer, either from friction in the machine or in the surrounding media, etc. To recognize and to

eliminate such factors has not been without difficulty. While one cannot say that all of these have now been got rid of, yet I feel assured that in the instrument I know best, the audion oscillator, we have been able to reduce to a minimum such factors as will greatly vitiate the results. Sufficient work has been done by independent observers which correlated with our own, enable one to present results with some assurance. With the machine at our disposal we have been able to approximate so closely to a measurement of the threshold that one can show curves which in the main outline the sensitivity of the ear at the pitches tested. It appears unnecessary to wait till the perfect machine arrives. Such a machine may be necessary for perfect physical measurement. Even with an instrument less perfect than ours, a mean can be obtained from the examination of several normal ears and the variations from the mean obtained by the same instrument under the same external conditions will be approximately correct for all ears so tested. Of course the more accurate the instrument the more accurate the information obtained.

Historical Part

In the past the physicist has largely worked in his special field undisturbed by any criticism or suggestion the otologist might offer. The otologist has not hesitated to acknowledge his great indebtedness to the physicist. It was the great physicist and physiologist, Helmholtz, who gave us the classical work on hearing. In 1862, Helmholtz published his "Sensations of Tone" and propounded his resonant theory of hearing—a theory modified by himself in later editions as new anatomic data were added, a theory which in spite of many objections still influences the work of physiologists and otologists, and in which the central idea of analysis of sound by resonance has the support of most of them. From the stimulus of that work most of our great aurists of the past drew inspiration, and on its results they explained many of their clinical findings. Helmholtz readily acknowledged and readily used in his

work the findings of the anatomist and naturalist. The problems he studied did not seem to require co-operation with an otologist. It is interesting here to note that the work now to be described has added one other and new objection to the resonant theory. This objection I shall briefly state in the words of my collaborator. "It was possible in the case of this patient to apply in the frequency region of 3,300 d. v. an energy 10 billion times as much as corresponds to normal hearing at this frequency without stimulating in the slightest degree the sensation of hearing in the region below 1,200 d. v. where the patient's hearing was practically normal. Now in the Helmholtz theory, pitch perception is due to the coincidence of the impressed frequency with the natural frequency of the fibers of varying length and tension which constitute the basilar membrane. It is, however, extremely difficult to see how a mechanical system such as that represented by the basilar membrane with its fibers and attached Corti's arches could possibly receive ten billion times the normal energy at a given frequency like 3,300 d. v. without imparting as much as one billionth of that energy to the adjacent fibers which corresponds to vibration frequencies below 1,200 d. v. If these latter fibers had in this case been thus forced into response with one billionth or even one hundred billionth of the incident energy, the patient would have had the sensation of hearing in the region below 1,200 d. v. If these considerations are fatal to the Helmholtz theory, there appears to be no alternative at present but to make pitch perception a property of the nerves themselves ,including their endings, without attempting to find a mechanical explanation. This amounts to nothing more than the assertion of our inability at present to obtain a satisfactory mechanical basis for pitch perception." John P. Minton, Ph. D., Physical Review, Vol. 19, No. 2, Feb. 1922, p. 95.

Though Helmholtz was not specially interested in the problem of the threshold of hearing, this phase of the subject attracted the interest of several of his successors. Very important work along this line was done by Lord Rayleigh.

To determine the threshold of hearing, for high notes he used a whistle mounted on a Wolfe bottle attached to which was a siphon manometer to regulate a constant air pressure. Having ascertained the distance at which a tone could be heard under as far as possible constant atmospheric conditions, and having calculated the energy emitted by the whistle from the pressure in blowing it, he was able to approximate to the amplitude required for the hearing of the tone. One serious objection was the distance the observer had to go from the source of the sound—in the case quoted 820 meters—involving the disturbing influence of atmospheric factors. To estimate the minimum audibility of low notes, Rayleigh used tuning forks which enabled him to approximate the sound to the ear. From the difference in the decay constants of the fork it was possible to calculate approximately the energy emitted. Rayleigh also used a telephone receiver as the source of sound. "The deflection of the diaphragm for a direct current was measured microscopically and this was considered the same as for alternating currents flowing through a receiver, currents having a period far below the natural period of the diaphragm. With the volume of air in the meatus known it was possible to calculate approximately the change in pressure in the ear drum from the current flowing through the receiver." In reading over Lord Rayleigh's work one readily appreciates the great advantage the audion oscillator offers for such an investigation and learns once more that advances in science depend to a large extent on improvement of instruments of attack.

The German physicist, Wien, investigated this problem by a different method and in 1889 published his results. He made use of the siren to estimate the minimum audibility of sound. The siren, one of the oldest instruments to produce a continuous series of sounds at different pitches, has holes at equidistance or teeth at equidistance. In the former, air is drawn through to produce sounds varying in pitch with the rapidity of vibration of perforated drums; in the latter, the electric siren, the make and break of a

current produces the tone varying in pitch with the rapidity of the make and break. Wien used the electric siren conveying the sound to the ear by means of a telephone. By assuming that the amplitude increases proportionately with the current he calculated the amplitude of the vibration for a tone at the threshold of audibility. His apparatus enabled him to observe results through a range of frequency from 50 to 15,000 cycles. As a result he placed the maximum sensitivity of the ear to sound at about 1,200 to 2,000 d. v., from which area the sensitivity dropped off rapidly on both sides. These results, extensively quoted by physiologists and otologists, especially in Germany, do not appear to be generally accepted by physicists. Two objections urged against the findings of Wien it is well for us now to keep in mind: (1) that the type of telephone receiver used introduces resonances affecting the findings of sensitivity in certain areas; (2) that the siren is not a suitable instrument to determine minimum audibility since the friction necessarily produced by its rotation introduces too many accessory sounds and further in the note of the siren overtones are prominent.

From this time on many physicists attacked the problem either by similar methods, eliminating objectional factors, or by other means; for instance, Kranz of the Sabine Laboratory used a thermal receiver as a source of sound. What concerns us more particularly is that only within the last few years do we find a systematic endeavor to get a closer co-operation of otologist and physicist. Even now many of the results published by physicists are based on the assumption that an ear which to the physicist appears to hear normally is therefore a normal ear, a dictum with which few of us will agree.

In the past there have been otologists with sufficient knowledge of the science of acoustics to appreciate the importance of the problems involved and with skill sufficient to attempt to express this threshold in physical measurements. For instance, Gradenigo attached to the upper end of a fork a dark triangle on a white background. When the fork is struck there is a double vision. One sees two pale triangles with a black tri-

angle common to both; the greater the amplitude the greater the space separating the two triangles; as the amplitude diminishes the two triangles merge more and more into the single black triangle. By noticing the extent of the field when the patient ceases to hear (marked by a millimeter scale), Gradenigo estimated the ratio of hearing. This method, however, is only applicable to low notes.

Cooperation of Otologist and Physicist Necessary.

My object in thus briefly sketching the work of the physicist has this for excuse, that I believe that at present and for some time to come the ascertaining of the threshold of hearing must be done primarily by a physicist but will have its best fruit when done in conjunction with an otologist. Few otologists have the necessary physical and mathematical training to understand the physical problems involved and to calculate the energies necessary to get correct results in estimating the minimum audibility of pure tones either in normal or defective ears, and this to my mind is at present the essential problem. Few of us have any conception of the infinitesimal amount of energy required for the ear to hear. Mr. Minton has calculated that the energy consumed by a 40 Watt lamp would operate a string of telephone receivers which if placed a foot apart, would encircle the globe 25,000 times and yet produce a tone of 1000 cycles that could be heard in each receiver if placed to an ear. The intensity of the sound transmitted to the ear is proportional to the energy. If the ear were a perfect physical instrument this would hold for the amount of energy transmitted to the nerve ending. But there does not seem to be the same relation between the amount of energy transmitted to the ear and that transmitted to the nerve endings; certainly in defective hearing this ratio is greatly altered at different pitches and to a varying degree.

Defects In Our Present Methods and Knowledge.

In spite of the large amount of work that has been done especially during the last 25 years to determine in absolute terms the minimum amount of sound, the amplitude of vibration that the ear can perceive, the resulting figures have been most dis-

crepant. The results of the various observers show a wide range of difference. One cause contributing to this was the inadequate apparatus available; another that no otological tests were made to ascertain if the ears so tested were normal; to these may be added that normal ears vary very considerably not only with the age of the patient but among individuals of approximately similar ages, and even between the ears of the same individual.

At present we depend almost entirely on tuning forks. The overtones in the lower forks are got rid of by clamps and in the higher forks are so much higher that they do not interfere with our present application of the test; we are also aided by the fact that the fundamental tone persists longer than the overtones. Tuning forks, however, have great limitations. They are wanting in scientific accuracy because we cannot well measure the amplitude of their vibration. Each vibration period varies in each set and alters somewhat with use. It has been impossible to give a percentage estimate of the impairment of hearing in one ear compared with normal by means of forks, for the amplitude of the tuning fork when it ceases to be heard in the two ears is difficult to estimate.

The otologist has at present no satisfactory means of measuring the intensity of the sound perceived. There is no certainty in regard to the position in the sound scale in which the maximum sensitivity for sound perception lies.

Our present tests with forks, with whistles, etc., give us a rough and ready method of ascertaining fairly accurately whether the lesion lies in the conducting mechanism of the middle ear or in the nerve including its endings. Such a wide generalization has served in the past, but leaves much that is important unanswered. There are few of us fully satisfied with the results obtained and most of us recognize the great gaps in the knowledge of the lesion. Our tests frequently fail us when we desire definitely to locate a lesion. Consider for a moment lesions in the conducting mechanism. Supposing the drum membrane be indrawn and have its curvative altered, we are not able to say how such an alteration will affect the hearing, whether the deficiency in hearing present be

due to this or to some added deficiency of movement of one or more ossicles. If the membrane be relaxed we are unable to state how much deficiency of hearing hereby results or the pitches involved. Such questions present themselves as what defaults in hearing are associated with perforations in various quadrants of the drum—quantitatively and qualitatively; in combinations of middle ear and nerve deafness, which factor predominates and to what degree. It has been stated that in the loosening of the malleus-incus point not only are the low notes diminished but also high notes of plus magnitude; the question arises what is the picture of ankylosis of these joints. But one need not stress this phase of the subject.

Method of Testing With the Audion Oscillator.

This method of testing the sensitivity of the ear or the degree of deafness consists, in its essential features, of measuring the minimum audibility current at various frequencies through the vibrations of the diaphragm of a telephone receiver adapted for this purpose. The intensity of the minimum audible sound acting on the ear is porportional to the square of this current. These minimum audible current readings are taken over a range of frequencies from 100 to 4,000 or 5,000 cycles, and the results plotted in the form of curves. Observations have been restricted chiefly to these ranges for two reasons: (1) They include the pitches most important for voice perception and (2) the telephone receiver specially constructed for this work was timed to a natural frequency of over 5,000 and so errors from this source were less likely to vitiate the findings.

The Audion Oscillator is used to supply the current to the telephone receiver, which is placed against the patient's ear at a constant pressure. The oscillator is set for the desired frequency and the current flows through the bridge circuit composed of non-inductive resistances, a, b, c, d. The resistances, c and d, are generally held constant while a and b are adjusted so that there is sufficient current through the receiver for the patient to hear without effort.

A short circuiting key when depressed, short circuits the receiver and when the patient wishes to listen he releases the key, listens very closely for a second or two and then depresses the key. The patient nods his head if he hears the tone in the receiver. To reduce the tone in the receiver are operator adjusts the resistances a and b and a slide wire resistance, so decreasing the current through the receiver. The patient again listens as before and indicates if he hears. In this way the current through the receiver can be continuously decreased until the minimum current to make the tone just audible to the patient is obtained: about the vanishing point of the tone the patient is required to listen intently for two or three seconds several times with two or three second intervals between each listening period in order to avoid fatigue. Having obtained the settings of the resistances for minimum audibility at various frequencies the current through the receiver is calculated by means of well known formulae from the values of the resistance of the bridge arms, the impedence of the telephone receiver and the total current indicated by a galvanometer. With these current readings and the vibrational characteristics of the special receiver, the vibrational energy at the various frequencies of the receiver diaphragm can be calculated. Taking the sound intensity as proportional to the values of the vibrational energy we can plot a curve showing the sensitivity of the ear at various frequencies where the sensitivity is considered to be equivalent to the reciprocal of the vibrational energy of the receiver diaphragm for minimum audibility. N. B.—A fuller description of the audion oscillator is being prepared and will be published separately.

To secure the exclusion of outside noises a sound-proof booth has been built in which the patient is seated during the testing. The booth is always used for testing normal ears and for testing ears which depart little from normal.

Method of Procedure

Previous to examination with the audion oscillator the

history of the patient is taken and details noted of previous trouble in the ear, nose and pharynx. The pharynx, naso-pharynx and nose are examined and the permeability of the tubes noted. The drum membranes are carefully observed and a record made of the Rinne and of the Weber tests and of bone conduction. The distance at which the whispered voice is heard is ascertained and fork tests are made to ascertain their diminution or increase from normal. In addition, in ears with defective hearing an examination of the tubes is made with the catheter and a careful examination of the naso-pharynx with mirror or naso-pharyngoscope. When indicated, X Ray plates have been made.

Lantern Slides to be Shown

In ears with normal hearing the curves are shown plotted in two ways: (1) In terms of relative receiver currents—relative to a mean of several normal ears. (2) In terms of the vibrational energy of the diaphragm.

In ears with defective hearing the curves are plotted relative to an average of several normal ears in terms of relative receiver currents.

SUMMARY OF RESULTS

I. Minimum Audibility In Normal Ears

The variety and irregularity of our results in ears with normal hearing were unexpected. You will have noted that while there exists a considerable variation among normal ears, each ear possesses one or more well defined peaks. The curves show these peaks of maximum sensitivity at various pitches but chiefly near 1,000 cycles. It will be noted that an ear may and usually does possess more than one well defined peak but that when two or more peaks are prominent it usually happens that one markedly overtops the other. While the maxima of sensitivity show great variety in pitch position in different individuals were one to indicate the position in the pitch scale where the maximum sensitivity lies in the average individual, one

would not go far astray if it were placed about 900 cycles.

It does not seem advisable to enter into details on this subject at this time; a complete report is being prepared and will be published later. It is to be understood that this subject is yet in the experimental stage and no deductions are offered. We do not know why certain ears have their maximum acuity as low as 500 or 600 cycles—others as high as 1,500 or 1,700 cycles. Age probably is a factor but it does not appear to be the only factor. While our greatest sensitivity was in a young man of 22, who at 900 cycles had a sensitivity of $1/750 \times 10^{10}$ ergs (an erg being the unit of mechanical energy), yet we found in a man of 35 years at 700 cycles had a sensitivity of $1/220 \times 10^{10}$ ergs. One interesting question for the otologist of the future will be the after history of ears with this extreme sensitivity.

II. Minimum Audibility In Ears With Defective Hearing.

In the immediate future it is the study of the curve of the abnormal ear that offers the most fruitful field and it is in this field that we as practical otologists are most interested. Glimpses of these possibilities have already been given in the papers of Dr. Dean and his collaborators and in the papers of Mr. Minton and myself. With the modified siren used by Dr. Dean and Mr. Budge, as well as with the audion oscillator used by Mr. Minton and myself, a mean audibility can be readily obtained in ears with normal hearing. In the results here shown a base line is drawn to indicate this mean and the values of the relative receiver current necessary to get the threshold at various pitches plotted relative to this, give a curve indicating the defects in hearing. A direct comparison of the sound intensity necessary for hearing in the abnormal ear compared with the normal can be got from the square of the relative receiver current, as the sound intensity is proportional to the vibrational energy of the diaphragm.

At present the curves from the Iowa laboratory and those from the Chicago laboratory are dissimilar. In considering

this question it must be remembered that a different method of plotting the curves has been used. In the papers issued from the Iowa laboratory the curves are plotted in numbers designated in terms of electrical resistance in the apparatus used and the ordinates are not proportional to the current. Our papers show the curves plotted in terms of relative receiver currents. The results should not vary greatly according to the machine used, though presumably they will vary somewhat according to the result obtained in normal hearing tests. But it is well to have at this stage a variety of testing apparatus; one day we will be able to decide which one is best suited for the purpose.

From our examination of curves obtained from ears with defective hearing we present no deductions. When we have sufficient data we shall begin to synthesise. Even now, however, the graphic pictures of some of these curves speak for themselves, for instance, the curves of nerve deafness. The complication of other curves as in otosclerosis make us hesitate to commit ourselves to any deductions.

The observations which I presented to you at our last meeting in regard to the position of tinnitus relative to nerve deafness have been confirmed by observations during the yast year—some of which have been shown. They throw an important light on the pathogenesis of tinnitus—at least in some ears.

One word in conclusion, we all recognize the amount of pessimism which exists not only among otologists but even among the general laity in regard to the outcome of the treatment of hearing defects. Such pessimism is not without cause. Yet this National Otological Society must be heartened by the character of this work being done not in one city but in various centres on this continent. The close co-operation between physicist and otologist is producing lasting results—results which can only be appreciated by one who has taken part in this co-operation. Such co-operation has come to stay and ought to be available to each of us. With the accumulated knowledge which will come, the cloud of pessimism which now oppresses us will be at least partly dissipated.

SUMMARY

I. An accurate means of ascertaining the minimum audibility of tone perception in ears with normal and defective hearing is needed in otology.

II. The results of testing by means of the audion oscillator surpass the means at present at our disposal.

III. By it a permanent record can be made of the amount of hearing present, of the progress of the disease and of the results of treatment.

IV. The method is full of promise for a more accurate diagnosis of the localization of lesions. It gives an explanation of tinnitus. This method offers a new route to investigate the physiology of hearing.

CERTAIN NEURO-OTOLOGIC PROBLEMS.

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Outline

I. Remarks about the new plan—the presentation of a broad subject, to which an entire session of the Society shall be given.

II. The scope and limitations of this paper. This paper is designed to include all the main subjects included under Neuro-Otology. The limitations of the paper. It will make no attempt to be encyclopedic; its point of view is purely clinical; certain experiments in physics and upon animals are included, but only to the extent that they throw light upon clinical problems.

III. Introductory remarks as to the part played by experimental research and clinico-pathologic research.

IV. Problems—taken up one by one as entirely separate entities, but conforming to a general plan.

V. Summary. An attempt to make the following classification, on the basis of the subject matter presented in this paper:

(*a*) What we are to regard as purely theoretic.

(*b*) What we are to regard as highly probable.

(*c*) What we can regard as reasonably proven—including the phenomena on which we feel we can rely in clinical diagnostic work.

I. Remarks About the New Plan of Presentation.

The council of the Otological Society considered that an experiment might be tried at this meeting of the Society. Instead of having a series of papers on different subjects, the council considered that our program would be improved if we concentrated our attention upon a few main subjects, devoting one session, or a large part of a session, to the discussion of a single subject. Further, that as far as possible, one member should be selected for preparing a formal presentation of the subject. It was further suggested that this paper be printed in advance of the meeting and copies put in the hands of all the members of the Society, so that they would come to the meeting prepared to add as much as possible to the discussion. In this way, it is hoped that such a paper, followed by a large and more or less prepared discussion by many members, might constitute a fairly elaborate symposium of this subject.

Such presentation and discussion of neuro-otologic studies seems opportune. There is no other phase of otologic work more in need of such a discussion than the problems of neuro-otology. The problem involved in the study of the internal ear and its intracranial connections is still in its infancy. On the one hand, very little has been definitely established; on the other hand, when we come to learn more and more definitely the significance of the phenomena that we actually see in the examination of patients, we can look forward to an enlarged sphere of the usefulness of otologic study. To be sure, at this date, our conception of neuro-otology is not so vague as it was only a few years ago. To those who have made such studies, it is encouraging to note that a certain semblance of order has already taken the place of what was so recently a complete chaos.

II. The Scope and Limitations of This Paper

This paper is designed to present all the main subjects included under Neuro-Otology. Broadly speaking, neuro-otology includes every investigation that bears in any way on the internal ear itself; the anatomic connections of the internal ear with the intracranial structures—in other words, the complete

auditory apparatus as well as the complete vestibular apparatus; the normal physiologic functioning of the auditory apparatus and the vestibular apparatus; and the phenomena observed in pathologic change in any portion of either the auditory or vestibular apparatus. In studying the literature, it is surprising to note how little advance has been made in the knowledge of the intracranial portion of the auditory apparatus. It is equally surprising, on the contrary, to note that there has been a remarkable advance in the study of the vestibular apparatus; this is especially true during the past five years. At the present time, as much as ten per cent. of all otologic literature seems to be made up of vestibular studies, by leading investigators, of many nationalities, notably the Dutch, Austrian, Italian, Belgian, French, Scandinavian, German, Japanese, English and American. Instead of occupying a very casual position in otology, vestibular studies now include so many investigators and such a mass of work as almost to constitute a specialty of its own.

For this reason, this paper will make no attempt to be encyclopedic or to refer to the extensive bibliography. It will attempt to present only the essentials of each main neuro-otologic subject. Its point of view is the purely clinical; certain experiments in physics and upon animals are included, but they were undertaken and are presented only in the attempt to throw light upon clinical problems that come up in usual otologic practice.

III. Introductory Remarks as to the Part Played By Experimental Research and Clinico-Pathologic Research.

The comprehensive neuro-otologic problem is to attain knowledge of the antomy and physiology of the auditory and vestibular mechanisms. The immediate anatomic problem is, through what structures, along what various pathways and to what neural termini, do the many and varied impulses proceed from the internal ear. The object of such study is to enable us to interpret the significance of pathologic disturbance of the various portions of each mechanism.

There are two main avenues of approach to this problem—the experimental and the clinico-pathologic. These two methods are essentially similar. Not only have they the same object to attain, but the underlying principles are identical, in that they both include the study of structures in relation to function, and the study of structures in relation to the loss or disturbance of function. They differ in that the experimental is largely confined to animals. It might be well to consider the advantages and also the limitations of each of these two methods of approach.

Experimental Research.

Experimental research has certain advantages over the clinico-pathologic, presenting as it does the opportunity either to stimulate or to destroy certain tissues, and then to study the results. These results can be studied by noting changes or loss of function, and also by alteration or impairment of reactions to stimuli. In addition, it is then possible, by post-morten examination, to make gross and histologic study of the structures affected. The further advantage of such experimental research is the opportunity afforded to establish a series of observations and to obtain a general average of results from many similar experiments.

Much important knowledge of the vestibular apparatus has already been attained by the experimental method. This includes observation and classification of the responses which can be obtained from a direct stimulation or destruction of separate structures within the internal ear. S. S. Maxwell has made recent valuable contributions to this phase of the subject, by experimental work on fishes; he will present a resumé of his work and that of others, in the discussion of this paper.

Another result of experimental work has been the tracing of degenerated nerve-fibers from the VIII Nerve and its connections, in various directions within the brain-stem and the cerebellum. The most recent and valuable work on this subject is that of Sachs and Alvis; Dr. Sachs will open the discussion by presenting the present status of available information from experimental work on animals.

There are many difficulties peculiar to vestibular study, that are met by one undertaking experiments upon animals. There is the difficulty of applying the principles of comparative anatomy and physiology; due consideration must be given to the variations of structure and function in the different animal species, including man. For example, the range and complexity of ocular movements varies greatly in different animals; vestibular stimulation which causes rotation of the human eyes in the frontal plane, produces in fishes merely an upward deviation of one eye and a downward deviation of the other. Caloric stimulation of cats fails to produce some of the responses with which we are all familiar in the human being.

One of the most reliable methods of tracing the course of nerve fibers is by means of the degeneration which follows their injury. Such degeneration can be traced only in the distribution of the particular fibers injured. Although, physiologically, nervous impulses pass from the terminals of one nerve fiber to another neuron, and so on to their ultimate destination, pathologically considered, each synaps limits further degeneration. To repeat, degenerations can only be traced within the limitations of a single neuron—always being limited by the synaps. Many different pathways, such as that of common sensation from the skin, are known to include three or more successive neurons, including as many synapses through which the impulse must pass. To follow this pathway by the process of tracing degenerations, it is necessary to produce a lesion in each successive neuron. For example, the sensory pathway for pain extends from the periphery to the posterior root ganglion; other fibers pass from there and enter the spinal cord; a short distance above the point of entry, connection is made with another neuron, the axis cylinder of which proceeds up the cord and through the brain-stem to the optic thalamus. Here in turn, new connections are made; the ultimate anatomic connections are as yet undetermined. The different forms of sensation, such as heat, cold, tactile, pressure and muscle-sense, are each represented by a special neural pathway which can be traced over successive neurons along its course. Some proceed up the spinal cord on the same side that they enter, some

on the opposite side; some are interrupted within the cord shortly after their entry, whereas others proceed without interruption to nuclei in the medulla oblongata.

Analogous arrangements may be noted in the nervous mechanism of the special senses. The retina, for example, contains within its layers not only the end-organs, represented by the rods and cones, but neurons interposed between these end-organs and the neurons which proceed in the optic nerve. Furthermore, the primary centers of vision form another interruption in the course of the visual pathways before they reach the cerebral cortex.

There are only a few demonstrated synapses along the vestibular pathways, such as Scarpa's ganglion and Deiters' Nucleus; however, it is fair to assume from the analgies just given, that there are numerous other breaks in the continuity of the neural vestibular pathways.

From the well established principles of secondary degenerations, it is readily seen that any part of any pathway can be traced only within the segment which has been injured, and that to trace any particular pathway throughout its various segments, it would be necessary to produce lesions such as to involve each segment in turn.

In applying this method to the present problem, there arises the practical difficulty of making discrete lesions. To be of value, experimental lesions must be limited so as to destroy a small amount of tissue, and must be so located as to produce the desired destruction without disturbing adjacent tissues. In vestibular studies, it would be highly desirable to produce such minute lesions within Deiter's, von Bechterew's and the Triangular Nuclei; the cerebellar nuclei, globosus, emboliformis, and festigii; the corpora quadrigemina and many other structures. The difficulties attending such attempts are obvious.

To give concrete applications of the above general principles, we might consider the possible results to be obtained by destruction of successive segments in the vestibular pathways—

(1) End-organ, (2) VIII Nerve and (3) Central Nuclei.

1. End-organ. The exact histologic course of fibers from each semicircular canal could be demonstrated if one canal could

be destroyed and its pathways traced. (The same could be applied to the utricle and saccule.)

It is possible to destroy one horizontal canal and its ampulla, in an animal previously proven to be normal by caloric stimulation. To prove that only the horizontal canal has been destroyed, it would be necessary subsequently to demonstrate normal function in the vertical canals. This proof is probably impossible because of the serious disturbance of the entire mechanism of the end-organ incident to the destruction of one canal. This would seem to be corroborated by the experiments of Sachs and Alvis, in the lack of uniformity of the spontaneous nystagmus which they observed, in the different animals in which only one canal had been destroyed.

Even if one canal could be destroyed and the fact of this isolated destruction proved by the caloric test, the secondary nerve degeneration does not extend throughout the physiologic distribution but stops by Scarpa's ganglion. Sachs and Alvis have demonstrated that destruction of all the canals is not followed by degeneration in the VIII Nerve.

2. The VIII Nerve can be destroyed and secondary degeneration traced to different portions of the brain-stem and cerebellum. However, it has not yet been possible to determine any relationship between any portion of these fibers and the individual semicircular canals, or the utricle or saccule.

3. Central Nuclei. Isolated destruction of the central nuclei is much more difficult; and yet, to correlate the different nuclei with the different portions of the end-organ, such a method seems to offer the only possibility for future study. In the work already done, in making discrete lesions in the nuclei of the brain-stem, so far as we know only the spontaneous phenomena have been observed. Additional information might be gained not merely by observing the spontaneous phenomena but by studying the responses to the caloric test.

If, for example, Deiter's Nucleus were destroyed and subsequently the horizontal canal failed to give normal responses whereas the vertical canals did produce normal responses, we could reasonably conclude that the fibers from the horizontal

canal go to Deiter's Nucleus and that the vertical canals fibres do not go to Deiter's Nucleus. So with each of the other vestibular nuclei—the von Bechterew's and Triangularis—if it ever becomes possible to make discrete lesions of these structures.

Summary: To find out the exact course of the fibers from the different semicircular canals appears to be impossible from experimental work on the end-organ or VIII Nerve. If ever this is to be attained, it seems that the only possibility consists in making discrete lesions in the brain-stem and then testing the different semicircular canals by the caloric test.

Clinico-Pathologic Research.

Clinico-pathologic study has its own peculiar advantages, among them:

1. The human being is the object of study. This eliminates certain fallacies always present in animal study. Studies of the human being are furthermore more complete, in that it is possible to secure the co-operation of the patient in making more elaborate tests than are possible in animals. There is another important advantage in that the human being can supply intelligent evidence of his subjective sensations, both spontaneous and after experimental tests.

2. Certain pathologic processes result in small discrete lesions, in locations within the central nervous system, which can not be duplicated by operative work on animals. This includes vascular lesions, such as thrombosis and small foci of encephalitis and multiple sclerosis. Incidentally, lesions of this type give more exact information than tumors or abscesses, in which pressure phenomena may obscure the picture.

3. Clinico-pathologic study has the advantage of having its workers constantly pressed by the need of accuracy in diagnosis. Every clinician feels the necessity of accurate interpretation of phenomena which he observed in every day practice. There may be, in the world, a dozen or a score of experimental investigators interested in neuro-otology. In contrast, every otologist and every neurologist is a potential investigator in clinico-

pathologic research. Furthermore, problems have been raised by clinical studies which have stimulated experimental anatomic studies. The ideal, of course, is to obtain exact histologic knowledge of each minute detail. In the meantime, the clinician can serve two purposes: first, to learn what he can from clinical cases, or operation or necropsy; and second, by his daily clinical observations to raise questions and present problems for the anatomist to try to solve.

In discussion experimental work on animals, it has already been mentioned that normal responses to ear-stimulation should be made a basis of observations. It may be said that clinical experience with human subjects has already fairly established the norm. We know, with hardly any reservation, that when any normal individual is turned or his ears douched, there is produced a definite eye movement of certain duration, in a definite plane; a definite vertigo of a certain duration, and a falling in a definite direction. If he be turned or douched to an extreme degree, he shows pallor, sweat and nausea.

With the above phenomena we become familiar, and all who undertake such tests become impressed with the uniformity of responses.

Each variation from the norm must receive due consideration. To be sure, a certain latitude must be given to individual variations within the norm; when, however, gross deviations from the norm present themselves, it may be concluded that some portion of the complicated mechanism has been disturbed. It might be well to cite briefly certain examples of clinico-pathologic experience in patients that proved to have intracranial lesions.

1. In one patient, ear stimulation produces normal responses from the horizontal canals and no responses from the vertical canals. Another patient shows the reverse—normal responses from the vertical canals and absent responses from the horizontal. Other cases, such as angle tumors, have shown no response from any canals of either ear, except one horizontal canal, and this showed entirely normal function. Two cases recently seen (Exhibit A and Exhibit B), not as yet pathologically verified, showed normal responses from all semicir-

cular canals, except one horizontal—the exact opposite of the phenomenon-complex of the angle tumor. We conclude that there is necessarily a neuraxial differentiation—that the fibers from the horizontal canal are sufficiently separated from those from the vertical canals to permit either to be involved without any involvement of the other. This will be taken up in detail under the separate "Problems".

2. Comes a patient who shows normal nystagmus, but impaired vertigo on stimulation of one horizontal canal. Another shows normal nystagmus and impaired vertigo from both horizontal canals. Another shows normal vertigo but impaired nystagmus. We are led to consider that from the horizontal canal, there is a further division of the pathways, on the one hand producing the nystagmus and on the other hand producing the vertigo.

Clinico-pathologic observations of lesions in different intracranial locations have shown such different phenomena that to explain them merely on the basis of pressure from internal hydrocephalus is untenable.

In spite of the limitations in experimental and in clinico-pathologic research, each method has its own peculiar advantages and in many ways supplements the other. Further progress will be determined largely by the measure of co-operation of the investigators using the different methods of approach.

IV. Problems.

To the clinician the problem is to determine the valuation to be placed upon each phenomenon observed in the study of patients. We will take up separately a number of debated subjects and will present, in skeleton form, certain evidence against and for previous conceptions of their significance. It will thus be possible for the discussion to round out our knowledge concerning these separate problems. Realizing that there are many doing neuro-otologic work that would not be present at this meeting, we also sent 80 questionnaires.

It will be noted that the subject of forced movements of the skeletal musculature is not included in these problems. Magnus

and de Kleijn and others have demonstrated physiologically that there is a lower circuit from the internal ear to the skeletal muscles—presumably via the vestibulo-spinal tracts. It would seem that these vestibular impulses are eventually distributed by spinal co-ordinating mechanisms in control of the musculature affected. In this sense, the vestibulo-spinal pathway is analogous to the vestibulo-ocular pathway. As to past-pointing, as tested by the Barany method, although it is essentially an adjustment to the conscious sense of movement, yet it is probable that tonetic influences through the lower pathways reinforce this adjustment. The falling response to ear stimulation appears to be essentially a forced movement, although there is a definite cerebral element. There is, however, so much uncertainty as to the clinical interpretation of forced movements that we have not attempted to present it as a problem.

The problems will be presented in the following order:

1. Auditory.
2. Endolymph movement.
3. Mechanism of ocular movement.
4. Concept of vestibulo-ocular pathways.
5. The quick component of nystagmus.
6. Spontaneous vertical nystagmus.
7. Perverted nystagmus.
8. Vestibulo-cerebello-cerebral tract.
9. Cerebello-pontine angle phenomenon-complex.
10. Neuraxial differentiation of pathways from the different semicircular canals.

Auditory.

This subject offers nothing in the form of an argument against or for any existing conceptions. The anatomy, physiology, pathology and symptomatology of the cochlea and the

auditory portion of the VIII Nerve are well established. The cortical cerebral areas for the reception of auditory impulses have been demonstated to be in the first temporal convolutions; this may be considered as having been rather conclusively determined, both by experimental work on monkeys and by clinico-pathological cases.

However, as to the course of the pathways between the cortical centres and the entrance of the VIII Nerve into the brain-stem, clinico-pathologic evidence is singularly meagre. A priori, it might be expected that various brain-stem lesions would exhibit an impairment of hearing. Observations of lesions within the brain-stem without hearing impairment are common. In fact, hearing impairment from brain-stem lesions is so uncommon as to be almost unknown in the experience of otologists. The generally accepted pathway of the auditory impulses is as follows: From the auditory nuclei by way of the lateral fillet to the corpora quadrigemina and internal geniculate in both direct and crossed relations; from this location the pathway continues to the first temporal convolution of each cerebral cortex. For many years no new evidence on this subject has been presented and we rely on the uncontradicted work of the older anatomists and pathologists. The explanation advanced for unimpaired hearing in cases of brain-stem lesions, is the neuraxial differention of the auditory fibres, by means of which each VIII Nerve is in relation with both sides of the brainstem. The auditory pathways on each side are made up of fibres in relation to each cochlea. In other words, with this arrangement of fibres, deafness in either ear cannot be produced by a lesion on either side of the brain-stem or cerebral pathways, but would have to involve these pathways on both sides. The absence of impairment of hearing from unilateral lesions of the supranuclear pathways is however noteworthy. Some evidence of impaired auditory function might well be expected in brain-stem lesions. It may be that our methods of testing auditory function might be improved in order that we might detect such a type of impairment. In the present status of our knowledge, however, auditory tests are of practically no value in giving evidence of lesions within the brain-stem.

Attention might be called to a separate neuro-mechanism concerned with the peripheral organ of hearing; this concerns the mechanism controlling the tensor tympani and stapedius muscles.

These two muscles are so arranged that they appear to be concerned with the adjustment of various degrees of tension of the drum membrane and the ossicles. These adjustments are made to accomodate the middle ear mechanism to various degrees of sound waves. The tensor tympani muscle is supplied by a branch of the motor division of the V Nerve; the stapedius muscles by a branch of the VII Nerve. In many respects the neuro-motor apparatus controlling the adjustment of these muscles is analagous to the accomodation mechanism of the eyes, in which the ciliary muscle innervated by the III Nerve, automatically controls the focus of the crystalline lens, to maintain clear vision.

So far as we know the action of these muscles is automatic. That there is a lower mechanism, presumably within the brain stem, and that a cerebral control can be exerted on this lower automatic mechanism is suggested by the following. One of the writers is able at will to produce a roaring in both ears. Try as he will, he cannot produce this roaring in only one ear; in fact he cannot make one ear roar louder than the other. This sound, produced by voluntary effort, consists of a rapid sequence of beats. There is a definite intermission between those beats. The beats and the intermissions are synchronous in both ears. There appear to be perhaps fifteen to the second; this is of course merely approximate, but there is a definite rythm. The slightest effort produces this roaring, at any time; it is however a definite effort to maintain the roaring. By concentrated effort the ears can be made to continue to roar for 18 seconds; the roaring then ceases, but can be made to recur at once after cessation. It would be difficult to explain this matter on any other basis than that of a voluntary control over the action of either the tensor tympani or the stapedius muscle, or both.

Endolymph Movement

The first problem of the vestibular apparatus is that of the

physics of the end-organ. Upon theoretical considerations it has been generally assumed and regarded as more or less determined, that the endolymph moves freely throughout all portions of the membranous labyrinth; that the endolymph movement in the semicircular canals acts upon the nerve receptors in the crista ampullaris of each canal; and that the direction of the endolymph movement determines the nature of the afferent impulse. The peculiar physical structure, and the configuration of the semicircular canals and utricle and saccule lends credence to this view. Given an organ made up of open tubes, placed in definite planes, and containing endolymph throughout their full extent, it has been thought obvious that this sense-organ registered changes of position of the head by means of mass movement of the contained fluid. Doubt has been expressed as to the accuracy of this theory, especially concerning the possibility of endolymph movement within the membranous semicircular canals.

Question:

Does the endolymph move freely within the membranous labyrinth?

Against:

Physical considerations.

1. It would seem that the small calibre of the membranous semicircular canals must interfere with mass movement of the lymph within them, on account of capillarity.

2. It would seem possible that pressure variations, without mass movement of endolymph, can explain the origin of impulses from the end-organs.

Physiologic considerations.

1. In fishes in which the semicircular canals and their ampullae had been destroyed, S. S. Maxwell has produced all normal eye responses, except the horizontal, by mechanical stimulation of the otoliths of the utricle. This is an important contribution to the physiology of the internal ear. He further showed that the horizontal canal can be ligated or elevated into the vertical position, without interfering with the responses

from turning in the horizontal plane. He points out that all the essential responses can be produced under conditions in which endolymph movement is impossible.

For:

Physical considerations.

1. The physical arrangement of the semicircular canals is theoretically ideal for recording inertia of the contained fluid, from every possible movement of the head.

2. Maier and Lion (Arch. f. Ohren, Nasen u. Kehlkopfhlk., 107:149, Leipsic, May 20, 1921) have observed movement of the endolymph within the semicircular canals of living animals, after caloric stimulation.

3. One of us has demonstrated mass movement of fluid within a capillary glass tube under conditions approximating those of the semicircular canals. The lumen of the tube is between .3 and .4 mm.; the tube is semicircular and both ends terminate in a small reservoir or artificial utricle. The entire apparatus is filled with vinegar, which contains particles in suspension, easily observed under the microscope. With this tube in a verticle position, and the reservoir uppermost, raising or reducing the temperature of one side is followed by circulation of the liquid which can be easily observed under the microscope. The movement of the liquid begins slowly and gradually increases to a maximum velocity which is maintained as long as the conditions of temperature in the tube are maintained. The movements of the liquid are always in the direction corresponding to the principles of the thermo-syphon. During one observation of these caloric tests, one end of the tube accidentally became blocked; when heat was applied near the opposite end, two currents in opposite directions were observed at the same point within the lumen of the tube. In other words, free fluid movement was demonstrated in two different directions within a tube of the calibre of .4 mm. or less.

The above physical experiment would seem to show that capillary resistance does not prevent mass movement of fluid within a capillary tube, when the fluid is in free communication with a common reservoir through both ends of the tube.

Physiologic considerations.

1. It is perhaps possible to explain Maxwell's experiments without sacrificing our faith in the old tenets of endolymph movement. He has emphasized the importance of the otoliths, about which so little is known; and he has shown that certain eye movements can be produced in fishes, irrespective of endolymph movement within the semicircular canals. This, however, is not positive evidence that, under normal conditions, the endolymph does not move.

The eye movements in fishes, upon which these observations are based, do not compare in variety or delicacy with the responses elicited in the human being. When the horizontal canal of the fish was ligated, it is possible that the impact of the fluid acted on the sensory nerve-endings in the ampulla, even though this movement could not continue throughout the entire course of the canal. A gross horizontal movement of the eyes could be obtained under these conditions; this, however is qualitative. and not quantitative. If after ligating the canal, there could be produced an after-turning nystagmus of normal duration and amplitude, and after the caloric test a full response of normal intensity and duration, we would then have more positive evidence against endolymph movement.

Again, when the horizontal canal was elevated to the vertical position, it is not probable that the ampulla was drawn out of place becuse it is directly adjacent to the utricle.

2. The argument that tension, and not endolymph movement, determines the response is not tenable; a change of tension without movement is impossible. If after turning, there is a tension at one end of the utricle and a relaxation at the other end of the utricle there must have been a displacement of the fluid content—in other words, mass movement.

3. In cases of fistula into a semiccircular canal, pressure applied produces *immediate* responses. These responses *cease promptly on the cessation of the pressure.* There is evidently no prolongation of the response after the stimulation has ceased. After turning, the nystagmus and vertigo normally continue from 20 to 30 seconds. From this it may be concluded that the stimulus has persisted from 20 to 30 seconds after the turning

has ceased. Immediately after turning, the nystagmus and vertigo are at their maximum. They gradually subside; the amplitude of the nystagmus becomes smaller and smaller and the individual feels that he is rotating slower and slower, until he comes to a full stop. This is a common observation and can be demonstrated on any normal individual; it would seem that it can only be interpreted on the basis of a movement of the endolymph, which is at its maximum immediately after the turning and which gradually diminishes until it comes to a full stop.

4. A recent pathologic case bears directly on this subject. (Exhibit C., Georgie Johnson).

In the examination of this patient, pneumatic pressure in the left ear produced:

1. Nystagmus, large amplitude, horizontal; slow component to the right; quick component to the left.

2. Past-pointing of all extremities to the right.

3. No falling—all responses were in the horizontal plane.

The nystagmus and past pointing appeared promptly when pressure was applied; continued while pressure was maintained; and ceased at once when pressure was discontinued.

These responses fulfill the conditions of the fistula test and indicate a fistulous opening into the left horizontal canal.

In contrast to the active responses from the fistula test, the left horizontal canal gave no response to turning or caloric stimulation.

Obviously the crista ampullaris of the left semicircular canal, with its sensory receptors, was functionating. The responses from its stimulation were prompt and qualitatively correct. they were elicited not only by air pressure, but also, as shown in the motion pictures, merely by touching the fistula region lightly with a cotton-tipped probe.

It is of particular interest that, although the crista ampullaris was actively functionating, yet it was entirely unaffected by turning and douching. Obviously, something interfered with the function of the horizontal canal itself. The most reason-

able explanation of these phenomena appears to be that there was an obstruction in the horizontal canal, posterior to the fistulous opening, which interfered with the endolymph movement within the canal, and which did not permit endolymph movement or impulse to affect the ampulla.

In Ewald's original experiment on pigeons, he plugged the horizontal canal just behind an opening in the canal. Pressure caused the eyes to rotate toward the opposite side. This case of Georgie Johnson closely resembles this experiment of Ewald. A similar case was shown by one of us before this Society in 1917—case of John Brooks. It happened that the fistula in this case was in the *right* ear and showed:

1. Nystagmus large amplitude, horizontal; slow component to the left; quick component to the right.

2. Past-pointing of all extremities to the left.

3. No falling—all responses were in the horizontal plane. These two cases, Georgie Johnson and John Brooks, show all the phenomena of horizontal canal stimulation, by means of the fistula test. Johnson showed the normal responses to be expected on stimulation from behind the left ampulla, whereas Brooks showed the diametrically opposite responses to be expected on stimulation from behind the right ampulla. No positive deductions should be made from either of these cases; the only positive proof would be complete histologic examination, demonstrating an obstruction in the horizontal canal, posterior to the fistulous opening. Short of histologic proof, however, these cases seem to be exactly analagous to the original Ewald experiment.

Resumé:

Considering the available evidence, the following conclusions are suggested:

1. Capillarity does not prevent endolymph movement within the semicircular canals.

2. Endolymph movement within the semicircular canals is an important factor in determining the character of the impulses from the end-organ.

3. The otoliths play an important part in the detection of movement; apparently the otoliths and cristae of the semicircular canals cooperate in function.

Mechanism of Extraocular Movement

The preceding problem concerned the physics of the end-organ and the mechanism of the production of stimuli; the next problem is that of eye movement resulting from ear stimuli. Before attempting, however, to consider the vestibulo-ocular mechanism, it seems wise to discuss the ocular mechanism with which the ear is connected. We will therefore attempt to analyze the essential mechanism of ocular movement itself; then to follow the functional relationship that exists between the internal ear mechanism and this ocular mechanism. Perhaps such a study of this functional relationship, correlated with historical experimental studies, may lead to an advanced conception of the anatomic relationship.

In the more primitive stages of the evolution of the ocular mechanism, one eye is placed on each side of the head and covers its own individual field of vision. In this stage each eye may be moved independently. In higher forms, the two eyes are placed anteriorly, and to a large extent cover the same visual field. The fields are thus necessarily superimposed, and fusion of images is attained. As binocular single vision, normal to higher animal types, necessitates maintaining fusion in all possible directions of gaze, an accurate adjustment of the visual axes must be maintained. Thus is evolved the conjugation of ocular movements—an automatic process. The term, "associated ocular movements," signifies the unity of purpose of the two eyes which operates in such a manner as constantly to direct both visual axes toward the fixation point in all possible directions of gaze. This association of movements is not only automatic and involuntary, but is so firmly established as to defy voluntary effort to avoid it. No normal individual can voluntarily rotate one eye in any direction in the slightest degree without also rotating the other.

By the very nature of the anatomic relations of the eyeballs and the extraocular muscles, it may readily be seen that *each of*

the twelve extraocular muscles must play a definite part in every *normal ocular movement.* In each movement, each muscle acts either as prime mover or synergist or control. These twelve muscles can instantly and with micrometric accuracy direct the two visual axes toward any point within the range of the possible eye movements. This is true not only as regards direction of gaze but also as regards distance. The accurate degree of convergence of the axes to correspond to the distance of the fixation point is a part of the adjustment.

What is the nervous mechanism involved in extraocular movements? Manifestly this involves at least three segments, viz: (1) The peripheral or neuro-muscular mechanism; (2) The cerebral projection tracts, and (3) An intermediate or coordinating mechanism.

1. The peripheral or neuro-muscular mechanism comprise the III, IV and VI Nerves and their nuclei, and the extraocular muscles which they supply. The nerves of this mechanism constitute the final common pathway for all impulses causing ocular movement. As previously indicated, this entire mechanism is called into action for each eye movement, whether voluntary or reflex.

Disturbance of any portion of this mechanism results in a porportionate loss of movement in terms of individual muscles or nerves, e. g. paralysis of the abducens, or paralysis of the III Nerve.

2. The cerebral projection pathway, by which voluntary ocular movement is accomplished, is commonly belived to be within the pyramidal tract.

It should be mentioned that fibers of the pyramidal tract have not been histologically traced directly to the cells of the eye-muscle nuclei. It should also be noted in this connection that pyramidal fibers have not been traced to the motor cells of the spinal cord, or indeed to any ultimate destination. From this we may gain some idea of the difficulties which beset histologic demonstration of a nerve pathway even though its existence be proven by physiologic observation.

From the viewpoint of the cerebral motor mechanism, the eye movements conform to the general rule applying to all bilaterally grouped voluntary movements. They are not paralyzed, and are but slightly affected by cerebral lesions which cause hemiplegia; either cerebral hemisphere is apparently capable alone of calling into action the complete range of normal extraocular movements. The usual interpretation assumes that there is a bilateral cortical representation for all muscles comprising such muscle-groups. Other examples of similarity grouped movements are: wrinkling of the forehead, phonation, respiration and trunk movements. All of these show but little involvement in the hemiplegic state.

Conjugate deviation of the eyes may result from acute unilateral cerebral lesions or irritation, usually in association with convulsions or disturbance of consciousness, but this conjugate deviation never persists as a permanent symptom. Cerebral lesions do not interrupt the conjugation of eye movements. This is true even following decerebration, if the mid-brain is not injured.

3. The coordinating mechanism of ocular movement. The peripheral neuro-muscular mechanism is well determined. The cerebral projection pathway is commonly accepted. However, to explain the phenomena of extraocular movement, it is necessary to postulate a coordinating neural mechanism, interposed between the cerebral and the peripheral segments. It is this coordinating mechanism that commands our particular attention. The intricacies of this structure have so far precluded exact knowledge, but its existence is proven by the following evidence.

In describing the phenomena observed in their experiments on dogs and cats, Wilson and Pike, (Phil. Tr. Roy. Soc. London 803:127-160, 1912) noted conjugate ocular movements produced by ear stimulation, (electric and caloric), in animals after decerebration. These eye movements were in lateral and oblique directions. Similar reflex conjugate movements were recorded after removal of the cerebellum.

In one of our experiments on cats, conjugate eye movements were observed after decerebration. A transverse incision was made anterior to the tentorium; necropsy showed that this incision had been carried to the base of the brain and that the cerebral hemispheres had been separated from the brain-stem, with exception of a part of each occipital lobe. Caloric stimulation of the right ear produced a few horizontal nystagmic eye movements; the amplitude was small and only a few movements were elicited, but the eye movements were definitely conjugate. After the head was removed from the body of the animal, electric stimulation, applied to the region of the internal ear of each side, caused conjugate vertical ocular movements.

The significance of these observations is in the persistence of the conjugation of eye movements after interruption of the cerebral or cerebellar connections. The type of movements observed requires the concerted, coordinated action of all the extraocular muscles. Since the cerebrum and cerebellum can be excluded, there remains only the brain-stem in which to look for this coordinating mechanism. It would seem that this mechanism must include a system of neurons with fibers distributed to all the cells of the peripheral motor nerves; also that this mechanism is capable of sending to all these cells, the correct impulses necessary to produce any accurately coordinated conjugate eye movement. This is also indicated by the variety of reflex eye movements observed after vestbular stimulation. It would seem probable that this coordinating mechanism not only receives afferent stimuli from various peripheral sources and directs the appropriate reflex responses, but that it is only by way of this mechanism that impulses reach the eye muscles, from the cerebrum or any other source.

Clinico-pathologic observations furnish important evidence concerning this mechanism. Many cases on record have shown inability to rotate the eyes in certain directions of gaze, although there was no paralysis of any of the extraocular muscles. These cases are designated as paraly-

sis of associated ocular movement to the right, or to the left, or upward, or downward, or in convergence. Such cases show lesions involving the posterior longitudinal bundle or the brain-stem near the aqueduct of sylvius. Cases of this type, showing a disturbance of associated eye movements without paralysis of any of the eye muscles, point definitely to lesions of the mechanism under discussion, and indicate that the posterior longitudinal bundles and other association and commisural tracts within the brain-stem are utilized by this mechanism.

According to the above conception, all eye movements, whether voluntary or reflex, are under the control of this coordinating mechanism. It would seem that this mechanism not only acts as a switchboard of distribution but also manifests a certain energy derived from its own neurons—similar to that of the electric amplifier. This energy is distributed in a selective manner and results in a conjugate movement. To cite an analogy in military manoeuvres: the two eyes, being analagous to a squad, execute "squads right" or "squads left" at the order of its leader; the leader moves his squad as a unit at the command of the company commander. If the commander is killed the leader still controls his squad as a unit. If no order be received by the leader, the squad may wander about aimlessly—still as a unit—and under the leader's direction. The squad leader in this analogy is the coordinating mechanism in the brain-stem.

The classic extraocular symptom of lesion of the peripheral neuro-muscular mechanism, is paralysis of individual ocular muscles; of the cerebrum, is conjugate deviation; of the coordinating mechanism of the brain-stem, is disturbance of associated eye movements.

Many other coordinated neuro-muscular mechanisms might be cited as analogies for this conception of a coordinating eye mechanism. For example, the respiratory movements are controlled by the respiratory center. All the movements of respiration are conducted by voluntary muscles, each supplied by peripheral motor nerves; the

whole series of these nerves arises from different levels, from the pons down to the lower dorsal segments of the cord. The respiratory center functions automatically through the influence of CO^2 in the blood; the automatic action of this center keeps up continuously throughout the life of the individual, whether asleep or awake. However, voluntary control through cerebral impulse, may step in and modify respiratory movement, by accentuation, change of rhythm or temporary arrest. Reflexly, various afferent impulses may also step in, such as application of sudden cold to the skin and emotional reactions. It must be noted, however, that whatever impulse is transmitted to the respiratory center, the response is in terms of synchronous action of many muscles controlling the respiratory movements. Decerebration or removal of cerebellum does not interfere with the automaticity of the respiratory mechanism, or with the harmonious action of the muscles it controls.

Coughing, sneezing and vomiting are controlled by similar coordinating mechanism or nervous centers each one of these centers serves its own definite purpose, and each center controls its own definite group of muscles to meet a specific physiologic need.

In this connection it is instructive to consider how few are the fibers of the cortico-spinal or pyramidal tract compared with the much greater number of peripheral motor neurons which they control. This signifies that each fiber of the cortico-spinal motor pathway controls a number of peripheral motor neurons through the intermediate neurons of a coordinating mechanism.

It must not be thought that the actual structure or content of these centers has been fully determined. The respiratory center is regarded as being situated in the medulla oblongata at the apex of the calamus scriptorious and is supposed to be comprised of cells located in the reticular formation on both sides of the midline. There is no unanimity of opinion, however, as to just what neurons are actually included in the formation of this center. The same

may be said for all the other coordinating centers. Their existence is unquestioned; their action, in a large measure an independent action, has been repeatedly demonstrated by experimental work as well as clinical observation; and yet we must not lose sight of the fact that their exact structure is unknown.

Summary:

Evidence indicating the existence of a coordinating mechanism, consisting of intermediate neurons situated between the cortico-medullary and peripheral motor pathway:

1. The complete conjugation of the eyes in all normal voluntary and reflex movements.

2. Conjugate ocular movements can be elicited by either cerebral hemisphere.

3. This conception of the coordinating ocular mechanism is in accord with other well-known coordinating mechanisms, such as the respiratory center.

Evidence indicating that this coordinating mechanism is located in the brain-stem:

1. Conjugate ocular movements can be elicited through vestibular stimulation, in animals, after removal of cerebrum or cerebellum.

2. Cases of paralysis of associated ocular movement have shown lesions in the brain-stem.

From the above consideration of the coordinating mechanism itself we now turn to a discussion of its functions; how it is brought into action by stimuli from the cerebrum and from various sense-organs.

Voluntary movements of the eyes, under normal conditions, consist of associated movements in all conceivable directions, except rotation in the frontal plane, and divergence. In close relation to the voluntary movements are certain instinctive automatic movements, in the production of which, a cerebral element is evident. Example: turning the eyes in the direction of objects suddenly coming into

the peripheral field of vision; maintaining fixation of vision upon moving objects; rotation of the eyes in the frontal plane, which normally occurs when the head is tilted to one side; the necessary degree of convergence to attain fusion for objects at different distances from the eyes; rotation of the eyes in the direction from which sounds are heard; or from which a touch is felt. This type of movement occurs instinctively, swiftly and accurately; without conscious effort, and occurs in the interest of securing visual efficiency. Conjugation of ocular movement is always maintained .

These instinctive automatic movements tend to approach the purely reflex. In fact it has been suggested by some that visual, auditory and tactile impresions may initiate ocular movements through the lower centers directly. There is some evidence that a direct reflex action can occur, but it is difficult to eliminate a cerebral element as a probable factor in most reflex ocular movements. There is, however, at our command one means of demonstrating a direct reflex movement of the eyes through the lower pathways; by vestibular stimulation in decerebrate animals.

Concept of the Vestibulo-Ocular Mechanism.

Up to this point we have discussed the essential mechanism of extraocular movement. We are now in position to discuss the functions of the mechanism in terms of its reactions to stimuli from the internal ear.

Regardless of the exact physics of endolymph movement, the eye movements resulting from the turning and caloric tests are always in the exact plane of the endolymph impulse; further, the direction of the slow component of the nystagmus is always in the direction of the endolymph impulse. The almost mathematical precision of these reactions is familiar to all observers, irrespective of what theory is advanced to explain them.

Electric stimulation of the end-organ, although a delicate test of function, is general and not selective. The caloric test is selective, but to a limited degree. The caloric effect

can be applied only from the lateral aspect; therefore the internal ear can not be influenced in sagittal or oblique planes. It is the turning test that not only involves both ears but stimulates them in a manner identical to their daily physiologic activity.

The turning tests can be made to influence the internal ears in any plane desired. In this way, an infinite gradation in the nature of the impulses can be sent inward from the end-organ, depending only on the position of the head during the turning. Each gradation calls forth its own specific movement of the eyes.

That a reflex associated movement of the eyes in any pre- determined direction or plane can be elicited, at will, by appropriate ear stimulation, implies an accuracy of adjustment and intimacy of neural connections of the utmost variety and nicety. We must postulate not only an accurate gradation of end-organ impulses, but a like series of adjustments of the ocular coordinating mechanism. In each adjustment, all twelve ocular muscles are concerned in an alternating series of actions and reactions.

Such phenomena can be explained only by the concept, that impulses from the internal ear go to the ocular coordinating mechanism, which determines the appropriate ocular movements. By this concept, fibers from the vestibular nerve and nuclei are not distributed to the peripheral motor neurons of the III, IV and VI cranial nuclei, but to the ocular coordinating mechanism. This would explain the failure of attempts to trace the vestibulo-ocular tracts to the eye-muscle nuclei, by means of secondary nerve-degenerations.

The Quick Component of Nystagmus.

Question:

Is it a cerebral function?

The usual concept of vestibular nystagmus has been that the quick component is of cerebral origin. There seems to be no question as to the origin of the slow component; but

there has been nothing conclusive concerning the origin of the quick component.

For:

The concept of the cerebral origin of the quick component is suggested by evidence such as

1. Voluntary fixation is a cerebral act. Deviation from the point of voluntary fixation occurs when the ears are stimulated. The explanation has been suggested that the quick component is an expression of the cerebral influence in momentarily overcoming the reflex pull from the ear.

2. During a vestibular nystagmus, gaze in direction of the quick component amplifies the nystagmus, whereas gaze in the direction of the slow component reduces it, even to the point of complete disappearance. It seems that it is optional with the cerebrum to determine a maximum or minimum nystagmus, by the simple expedient of voluntarily reinforcing either the quick or the slow component.

3. Loss of the quick component under general anesthesia; also under other unconscious states, such as coma.

4. Case of Miss C. (Equilibrium and Vertigo, P. 382). On caloric stimulation of right vertical canals, there occurred a rotary conjugate roll of both eyes toward the right, no quick component to the left; stimulation of right horizontal canal produced conjugate deviation to the right, which persisted for the duration of the stimulus without any quick component to the left. Stimulation of the left vertical canals produced a normal rotary nystagmus to the right. Stimulation of the left horizontal canal produced normal horizontal nystagmus to the right. The contrast was definite; the quick recoil to right was present and the quick recoil to the left was absent. Necropsy a few days later showed a large subcortical abscess in the right parieal region.

Against:

In contrast to the above, the following arguments make it strongly suggestive that the quick component can be

initiated by a lower mechanism, independent of the cerebrum.

1. The first argument given above, (that the quick component is an attempt at cerebral fixation), is minimized when we note that the quick component is no less active when visual fixation is eliminated—by closing the eyes. Further the intimate reciprocal relationship between the slow and quick components would seem to indicate the operation of a single mechanism, rather than the cooperation of widely separated structures. On the basis of a cerebral origin of the quick component, we would have to consider the following sequence; ear stimulation would act for the fraction of a second, whereupon a cerebral impulse would inhibit that from the ear and then substitute one of its own, effecting a reverse movement. This cerebral impulse, quickly subsiding, is then replaced by that from the ear and this alternating series of reverse impulses continues for the duration of the stimulus from the ear. This appears less reasonable than a concept of the operation of a lower coordinating mechanism.

2. Absence of the quick component in general anesthsia or other unconscious states by no means proves its cerebral origin. The different degrees of disturbed consciousness are accompanied by the loss not only of cerebral activities, but also by the loss of the various lower reflexes at different stages of the anesthesia.

3. Numerous cases of lesions in the cerebrum, including hemiplegias, temporal lobe lesions and occipital lobe lesions have failed to show loss of quick component.

4. Many cases of proven posterior fossa lesion, including cerebellar tumors and lesions of the brain-stem have shown absence of quick component. (Cite case of cerebellar abscess—Castillo boy, Exhibit D.)

5. Certain cases of pathology of the brain-stem in which ear stimulation produced dissociated nystagmus—nystag-

mus of one type in one eye and of a different type in the other eye. It would be difficult to explain these two different types of quick components on a cerebral basis. When one sees, after ear stimulation, a horizontal nystagmus in one eye and a vertical nystagmus in the other eye, it seems more reasonable to conclude that the quick component originates in a lower mechanism which automatically reacts to correct the particular deviation of each eye.

6. This conception of a lower automatic center which can produce the quick component, independent of the cerebrum, seems to be supported by recognized analogous coordinated reflexes. If acid is applied to the side of a decerebrate frog, the hind leg of the same side is moved in a manner to wipe off the irritant. This movement is not simply a flexion or an extension of the leg, but an orderly sequence of movements including flexion and extension of several joints in a manner to accomplish a definite purpose. If the back of a decerebrate dog is rubbed, the so-called "scratch reflex" may be elicited, consisting of a series of quick to and fro scratching movements of one hind leg. If a stream of cold water is directed against the inner surface of the pinna of a decerebrate cat, one may observe a quick shaking to and fro movement of the head which is characteristic of normal cats in attempting to dislodge irritants from the ear.

In an analysis of the movements concerned in such coordinating reflexes in decerebrate animals, it is particularly to be noted that there is in each reflex a series of reciprocating to and fro movements, performed by groups of muscles. Many muscles are concerned in each reflex, and different groups are active in different phases of the movements, alternating with remarkable accuracy and rhythm. These reflexes are of the nature of purposeful movements, and each of them can be initiated by volition. They have probably evolved from purely voluntary acts to the stage of automaticity of the low coordinating centers. As in vestibular nystagmus, some of these reflexes exhibit a series of to and fro movements, consisting of a principal movement

and then a movement of recovery, in preparation for a repetition of the principal movement.

7. Some direct evidence has been presented from experiments on animals, indicating that lower centers may initiate the quick component of vestibular nystagmus. Barenne, cited by Wilbrant and Sanger (Neurologie des Auges, achter Bond, Die Bewegungssorungen der Augenmuskeln, P. 308) observed no disturbance of nystagmus from turning in either direction, nor from caloric stimulations of either ear, in rabbits after removal of one cerebral hemisphere. Wilson and Pike in 1911 observed spontaneous labyrinthine nystagmus in a cat after destruction of the left labyrinth and the removal of the cerebral cortex of both sides. The optic thalami were intact in this experiment.

One of our experiments, (previously mentioned) although by no means conclusive, was not without significance. A cat under ether, was decerebrated to the extent that most of the cerebrum was separated from connection with the brain-stem. This was accomplished by an incision parallel and 1 c. m. anterior to the tentorium pasing through the optic thalami. In spite of surgical shock from which the animal did not recover, a few movements of both slow and quick components of nystagmus were elicited by caloric stimulation of the labyrinth.

Resumé:

The quick component of vestibular nystagmus is initiated by the lower automatic mechanism, independently of the cerebrum. However, cerebral impulses can modify the action of the lower mechanism.

Spontaneous Vertical Nystagmus

Question:

Is spontaneous vertical nystagmus indicative of lesion within the brain-stem, or involving the brain-stem by adjacent pressure?

Against:

1. A small nystagmus in extreme position of lateral

gaze is recognized as physiologic. Why should we not also regard as physiologic a small vertical nystagmus, in the extreme positions, upward or downward, of vertical gaze?

2. Observations on normal individuals have demonstrated a small vertical nystagmus upwards, after prolonged fixation in the extreme upward direction.

3. In patients showing general neuro-muscular exhaustion, we have frequently observed prompt vertical nystagmus of fair amplitude, on extreme gaze, upward or downward.

4. Active vertical nystagmus, with the quick component upward, was observed by us in a cat upon recovery from ether, after injury of both internal ears. Necropsy showed that there was no intracranial injury.

For:

1. Patients showing general neuro-muscular exhaustion, without evidence of brain-stem lesion, have shown not only vertical nystagmus, but also horizontal, oblique and convergent nystagmus depending upon the direction of gaze.

2. Clinically, labyrinthitis produces a spontaneous horizontal nystagmus or rotary nystagmus, or, most frequently, a mixed horizontal and rotary nystagmus. We know of no case of proven end-organ lesion which has shown spontaneous vertical nystagmus, with the exception of the cat experiment noted above.

3. Brain-stem lesions frequently exhibit spontaneous vertical nystagmus.

4. Cerebellar lesions, angle tumors and other lesions in the posterior fossa, frequently show spontaneous vertical nystagmus. In our experience, vertical nystagmus has been observed in cases of posterior fossa lesion of the type of tumor or abscess—lesions producing local pressure. Others have made similar observations. However, that

these lesions produce nystagmus not intrinsically but because of pressure, has not been regarded as conclusive. A recent case, (Exhibit D.), is illuminating on this point. The case was one of large cerebellar abscess. Among other phenomena, there was a large spontaneous vertical nystagmus upward. The abscess was drained. After operation the vertical nystagmus was absent. Later, autopsy showed that the abscess was limited to the cerebellum. This case suggests that it was not the intrinsic cerebellar involvement that produced the nystagmus, but the pressure upon the brain-stem.

Resumé:

Spontaneous vertical nystagmus, per se, is not necessarily indicative of brain-stem lesion, for the reason that it has been observed as a fatigue symptom. However, spontaneous vertical nystagmus has frequently been observed in patients with lesions in or near the brain-stem and, so far as we know, has not been observed clinically in association with lesions in any other location. In patients who have other symptoms of intracranial lesion, a spontaneous vertical nystagmus indicates that the lesion is within or adjacent to the brain-stem.

Perverted Nystagmus

Question:

Is perverted nystagmus indicative of lesion within the brain-stem or involving the brain-stem by adjacent pressure?

Definition:

We consider this phase of the subject of sufficient importance to describe the different types of abnormal eye responses to ear-stimulation, and to attempt a rough classification of perverted nystagmus.

1. Cases showing normal type of responses from stimualtion of one ear, but abnormal responses from stimulation of the other ear.

2. Cases showing abnormal responses from stimulation of both ears.

3. Simple perverted nystagmus—in which the eyes are coordinated, but the response is of a wrong type. Examples:—horizontal canal producing rotary, oblique or vertical nystagmus; or vertical canals producing horizontal, oblique or vertical nystagmus, instead of the normal rotary.

4. Inverse nystagmus, in which the nystagmus is diametrically opposite to the normal response.

5. Convergent nystagmus, in which each eye shows nystagmus inward.

6. Dissociated nystagmus, in which the nystagmus of one eye is of a different type from that of the other.

Against:

1. One should be guarded in pronouncing that a slight deviation from the normal type of nystagmus after stimulation is a true perverted nystagmus, because of the possible fallacy of imperfect technique. If one observes a rotary element in the response, after turning with the head 30 degrees forward, it would be wrong to draw hard and fast conclusions that this is a perverted nystagmus. Similarly, after douching with the head back 60 degrees, if one observe a rotary element, he must be sure to note whether the backward inclination of the head is at exactly the correct angle. It is also conceivable that the semicircular canals are not always in exactly the same plane in all individuals, as all physical structures occasionally show variation from the average normal. If, therefore on stimulation of the horizontal canal one observes a rotary element or a nystagmus in a slightly oblique plane, a certain latitude of interpretation is necessary. The abnormalities should not be considered as a true perverted nystagmus.

2. At each synaps in the course of nerve pathways, there is probably a regrouping of the functional relations

of the nerve fibers and a possibility of diversion of the pathways along which the impulse may pass. So long as an impulse is transversing one fiber, its course is fixed; at the synaps, there may be several avenues over each or all of which the impulse may continue. Every synaps in the course of the pathway increases the complexity of the pathways open to the impulse. Theoretically, if there is a block in any one segment of a pathway made up of several neurons, the impulse may deviate from its usual course by overcoming the threshold of collateral connections. One such a theory, Scarpa's ganglion might be the location where a lesion would divert the impulse from the horizontal canal to the pathways of the vertical canals. If this were true, it would be possible to account for a perverted nystagmus from a lesion not in the brain-stem, but within the internal auditory canal.

3. Even if the evidence were conclusive that perverted nystagmus is always of intracranial origin, indisputable evidence must be produced before we should state that it is indicative of brain-stem involvement. Supratentorial lesions, merely by increasing general intracranial pressure, may cause various localized phenomena, such as papilledema or abducens paralysis. It is conceivable that such general pressure might so derange the vestibulo-ocular mechanism as to produce perverted responses.

4. In the absence of conclusive data as to the exact mechanism involved in the production of perverted nystagmus, and in the absence of a great number of well-recorded and proven cases, it is unwise to regard this phenomenon as a positive indication of brain-stem involvement. To prove that it is not, all that would be required would be one case of frontal lobe tumor (or any other lesion at a distance from the brain-stem), which exhibited perverted nystagmus of any type.

For:

1. From our knowledge of the mechanics of the end-

organ and our interpretation of the normal physiologic responses, a perverted nystagmus is theoretically inconsistent with any disturbance of the end-organ or VIII Nerve.

2. The reflex pathway for normal nystagmus is from the vestibular end-organ, through the VIII Nerve and brain-stem to the ocular-motor nuclei. Theoretically it would appear that perversion of the normal response must be due to a disturbance along this pathway; if the end-organ and VIII Nerve can be excluded, this disturbance would appear to be necessarily within the brain-stem.

3. We know of no proven case of end-organ lesion which has shown perverted nystagmus. Neither do we know of any case of neuritis of the VIII Nerve which has exhibited this phenomenon. Tumors of the VIII Nerve frequently exhibit perverted nystagmus; here however, we have not only VIII Nerve lesion but a tumor mass distorting the brain-stem by pressure. It is a significant observation that in the tumors of the VIII Nerve that we have seen, perverted nystagmus never appeared as an early sympton but came on later, presumably in the course of the enlargement of the growth.

4. Lesions of the brain-stem itself frequently exhibit perverted nystagmus.

5. Lesions adjacent to the brain-stem of the type causing pressure, frequently show perverted nystagmus. It is sometimes noted, (as in case of Mrs. Fernandez, Exhibit E.), that perverted nystagmus comes on in the course of the development of a posterior fossa lesion; as the lesion progresses the nystagmus becomes perverted.

6. We have no record of any proven case of lesion not adjacent to brain-stem, which did exhibit perverted nystagmus.

Resumé:

Although this phenomenon has been observed for only a few years, and although the number of proven cases is

limited, yet it may be said that a true perverted nystagmus bids fair to be regarded as one of the most reliable signs of an involvement of the brain-stem.

Vestibulo-Cerebello-Cerebral Tract

Before considering whether the impulses producing physiologic vestibular vertigo pass through the cerebellum, we should first consider the evidence from which we assume the existence of a pathway from the vestibular nuclei to the cerebrum. Everyone recognizes that the appreciation of movement is a concept that may be derived from various sensations, including visceral, tactile, muscle and vestibular sense. The full normal recognition of movement is unquestionably a composite of sensations. In this composite of sensations, the element that we are discussing is the vestibular sense. This, we regard as a specific special sense.

We observe that from each type of stimulation of the end-organ, affecting the internal ear in various planes and directions, there occurs, in consciousness, a specific sense of motion. This sensation is definite in regard to direction, plane, duration, intensity and rate of movement—always corresponding to the character of the stimulation employed. To our minds, this is proof that each impulse has reached the sensorium.

It is generally accepted that there is a cerebral center for each of the other senses; it would seem reasonable to postulate a similar cerebral representation for the reception of impulses from this end-organ of special sense. As to the location of such a center, there is little definite evidence. Certain evidence, however, has been presented by Dana, Mills and Cushing, suggesting that the temporal lobe may be the location of this center. Two cases of right temporal lobe lesions were reported by Dana; one had exhibited vertigo, and the other, vertigo and forced movements, backwards and to the right. Mills observed a case of tumor of the left mid-temporal region, in which vertigo was an early symptom and had persisted for five years. Cushing has noted the frequent presence of nystagmus, vertigo and incoordination in patients with temporal lobe lesions.

Whatever its location, this vestibular center is to be regarded

simply as the cerebral terminus for the reception of vestibular impulses—hence a *sensory center*. It is not an "equilibratory center." Equilibration is a complex motor process. (Exhibit F., moving pictures of cats.)

Question:

Do the impulses from the internal ear, producing a sense of movement, pass through the cerebellum en route to the cerebrum?

Against:

1. So far as known, none of the other sensory pathways pass through the cerebellum. The visual and olfactory pathways enter the cerebrum above the brain-stem. Those of tactile, heat, cold, pain, pressure, kinesthetic, auditory and gustatory sensations, enter the cerebrum directly by way of the brain-stem. Lesions of the cerebellum are never characterized by any loss or any impairment of these sensations. If, as expressed above we consider that the impulses producing a sense of movement reach the cerebrum, from analogy of the other sensory pathways it would seem improbable that the vestibular impulses traverse the cerebellum.

2. Although fibres have been traced from the vestibular Nerve into the cerebellum, these may be accounted for as pathways concerned with functions not reaching consciousness. We have an analogy in the direct cerebellar tracts of the spinal cord, which enter the cerebellum via the inferior peduncles. according to the generally accepted view, these tracts, although they carry afferent impulses, are not concerned with sensations in consciousness. It would seem probable that the vestibulo-cerebellar fibres, like the direct cerebellar tracts, do not convey impulses concerned with the sense of movement, but constitute the afferent pathways for impulses to the cerebellum, resulting in coordinations of an automatic nature.

In consideration of these analogies, it might be inferred that the pathways conveying the impulses for the production of conscious sensations of motion from vestibular stimulation probably pass through the brain-stem, without any cerebellar connections.

For:

1. As before stated, fibres have been demonstrated to continue from the vestibular nerve into the cerebellum. Fibers have also been traced from the cerebellum into the cerebrum. These fibers traverse the superior cerebellar peduncles and decussate in the brachium conjunctivum. This arrangement would seem to furnish an anatomic basis for the theory that vestibular impulses pass through the cerebellum en route to the cerebrum.

2. Each case of proven cerebellar lesion that we have examined, has shown impaired or absent sensory responses from ear-stimulation. The experience of many others has corroborated this observation.

Resumé:

It would seem unquestioned that motion-sense impulses from the ear do reach the cerebral cortex. The location of the cortical center is not known; the only evidence that has been presented indicates its location in the temporal lobe. Although it is by no means proven, such evidence as is available suggests that these impulses traverse the cerebellum enroute to the cerebrum.

Cerebello-Pontile Angle Tumor Phenomenon-Complex

During the past six years we have observed the following phenomena in cases of angle tumor:

1. On the side of the tumor, a loss of auditory and vestibular function.

2. On the opposite side, normal auditory function, normal responses from the horizontal canal, but absence of responses from the vertical canals.

In brief, neuro-otologic examination showed a loss of function on both sides except the cochlea and horizontal canal of the ear opposite the tumor.

In some cases, during the early period of development of the tumor, we noted merely the auditory and vestibular loss of function on the same side. In the course of weeks or months,

one or more of the following additional phenomena were elicited:

1. The impairment and later, loss of responses from the vertical canals on the opposite side.

2. A spontaneous vertical nystagmus has been frequently observed. In certain cases, this vertical nystagmus was not observed on first examination but appeared after an interval. After such a case has once shown spontane-vertical nystagmus, we have never observed it to disappear, although we have noted variations in the amplitude of this verticular nystagmus.

3. In some cases, which had shown the complete phenomenon-complex, the horizontal canal on the opposite side had produced entirely normal nystagmus, vertigo and past-pointing. In other cases, during the early period of development of the tumor, the horizontal canal of the opposite side has shown normal nystagmus, but after an interval has produced perverted nystagmus.

4. In cases of well-advanced growth in the angle, we have observed abnormalities in past-pointing. This has been observed in a spontaneous past-pointing, and also in an absence or the impairment of past-pointing after ear-stimulation. In some cases, we have noted that we have termed a "crossed past-pointing;" both arms would past-point inward, or, both arms would past-point outward or, both arms would past-point outward, regardless of the type of stimulation. For example, the patient would past-point inward with both arms, regardless of whether he had been turned to the right or to the left.

It is difficult to interpret this phenomenon. In fact, our own experience has led us to draw no conclusions on the basis of abnormal past-pointing alone. Abnormalities in past-pointing have been observed in cases of cerebral lesion, as well as lesions within the posterior fossa; furthermore, we observed normal past-pointing in a patient who proved to have a cerebellar tumor. Although abnormalities in past-pointing do not appear, per se, to have anything approaching an exact significance or to furnish direct evidence in intracranial localization,

it would seem wise, however, to make careful tests of the past-pointing in all intracranial cases; abnormalities in past-pointing can at times be of distant value when correlated with other phenomena.

This phenomenon-complex has been observed and corroborated by many others in this country. However, we have no knowledge of its recognition in other countries. The only explanation of this phenomenon-complex would appear to be as follows:

1. The tumor destroys the function of the VIII Nerve on the same side by direct involvement of the VIII Nerve.

2. The isolated loss of responses from the vertical canals of the opposite side; the occurrence of spontaneous vertical nystagmus; the occurrence of perverted nystagmus on stimulation of the opposite horizontal canal; the abnormalties of past-pointing —all of these phenomena would appear to be caused by pressure of the tumor upon the pons.

Cautions:

Experience has demonstrated the need of caution in drawing conclusions in cases of suspected angle tumors. The number of cases of cerebello-pontile angle tumors, carefully studied by neuro-otologic tests, is insufficient to establish a complete working basis for interpretations.

We have recently studied a case, (Jesse Smith, Exhibit G.), which shows that other pathology than tumor can produce certain otologic phenomena of angle lesion. Jesse Smith showed involvement of the III, IV, V, VII and VIII Nerves, all on the right side. Ear tests showed complete loss of function in the right ear—both auditory and vestibular. Five days after the first examination, auditory function began to return; within two weeks, under our direct observation, instead of complete deafness in the right ear, there was such a complete restoration of hearing that he was able to hear a faint whisper, with the noise apparatus in the left ear. The caloric test also demonstrated the return of vestibular function. The vestibular function also returned within a period of two weeks. The facial paralysis also disappeared. The neurologic diagnosis was mul-

tiple sclerosis. In this case it is to be noted, however, that there was no impairment of responses from the vertical canals of the opposite side.

In the earlier stages of angle tumors, the phenomenon-complex is incomplete. Most assuredly, a diagnosis of angle tumor is not to be made, based only upon abnormal or absent responses from the one ear. Given, however, a picture of absent auditory and vestibular function in one ear; impairment or perversion of responses from the opposite ear, with normal auditory function in the opposite ear—we have data indicating involvement of the angle.

Even under these circumstances, however, one cannot state that there exists a tumor which is confined to the VIII Nerve and is probably removable. We have seen this phenomenon-complex in tumor of the pons and tumor of the lateral cerebellar hemisphere, invading the angle.

It is occasionally possible to draw valuable inferences from the history of such a case. If, over a considerable period, all the symptoms have been of the type referable to VIII Nerve involvement, and then there begin to appear the absence of vertical canals responses on the opposite side and other phenomena of brain-stem involvement, we are justified in stating that the evidence makes probable a true neuroma of the VIII Nerve. (Miss Buchart, Exhibit H).

The first symptoms of angle tumor are frequently those referable to functions of the VIII Nerve; often it is the otologist who is first consulted by patients with this lesion. While the diagnosis of angle tumor is not dependent upon neuro-otologic information, this phenomenon-complex has been observed so frequently in proven cases that its presence may now be regarded as a valuable diagnostic sign.

Neuraxial Differentiation of the Fibres From the Horizontal Canal and the Fibres from the Vertical Canals.

Before discussing this problem, it is necessary to state exactly what is meant by this neuraxial differentiation. Our con-

ception of this differentiation is that there is a divergence of vestibular fibres within the brain-stem, in such a manner that the fibres in relation to the vertical canals are separated by appreciable distance from those in relation to the horizontal canal.

This separation is considered to be sufficient to permit macroscopic lesions to exert a selective effect upon one set of fibres and not upon the other.

Here it should be noted that the various vestibular responses have generally been described in terms of the semicircular canals, without recognition of the probable cooperation of the otolith organs of the utricle and the saccule in the production of these responses. The work of Maxwell has made clear that the utricle is a definite factor in the production of reflex ocular movements; we should always bear in mind the probability of the participation of the otoliths of the utricle and saccule, to a greater or less extent, in the registration of movement in any plane. Therefore, although we are unable to estimate the exact relationship of the different nerve receptors, and consequently, for convenience, speak in terms of the semicircular canals, yet we should not lose sight of the part that may be played by the otolith organs.

Question:

After entering the brain stem, do the fibres from the horizontal canal pursue a different course from those conveying impulses from the vertical canals?

Against:

1. No histologic proof of these separate pathways has been furnished. There are many vestibular pathways which have been traced histologically:

(*a*) Fibers from the vestibular portion of the VIII Nerve have been traced to the so-called vestibular nuclei in the brain-stem—the Deiters', Bechertow and Triangular Nuclei. These nuclei occupy an area of considerable size and extent throughout the lower portion of the pons and the upper portion of the medulla oblongata.

(*b*) Fibers from the vestibular portion of the VIII Nerve have been traced into the cerebellum without interruption.

(*c*) Fibers from the vestibular nuclei, by association and commisural pathways, have been traced in various directions, to the posterior longitudinal bundles, into the spinal cord and the corpora quadrigemina, of both the same and opposite sides.

All that is known of these nuclei and fiber tracts within the brain-stem and cerebellum, is that they bear some relation to the vestibular portion of the VIII Nerve. No nucleus or separate fiber tract has been identified as being in relation with the utricle or saccule or any semicircular canal.

2. Funtional differentiation of fibers does not necessarily imply a divergence of their anatomic course. Pathologic processes may involve certain fibers, while others in close proximity escape. This may occur even within a nerve trunk, as in degeneration of the papillo-muscular bundle of the optic nerve. The destruction of this particular bundle of fibers within the optic nerve has been histologically demonstrated in patients who have shown blindness of the central portion of the visual field. A similar selective process may occur within the VIII Nerve; it is not a rare observation that in a neuritis of the VIII Nerve, as in syphilis, the cochlear portion may be affected and the vestibular portion not affected, and vice versa. It requires no stress of the imagination to conceive that within the VIII Nerve there may be an involvement of the particular fibers in relation to one semicircular canal, without the involvement of the rest of the nerve. Similarly, at any point throughout their intracranial course, it would seem that a pathologic process might affect certain fibers without affecting those even immediately adjacent.

3. The only evidence in favor of separate pathways from the different canals has been the clinico-pathologic. In the experience of all who have made vestibular tests of intracranial cases, it has been the vertical canals that have

shown impairment far more than the horizontal canals. The non-response of the vertical canals should not be regarded as conclusive evidence that the vertical canals fibers are widely separated from the horizontal canal fibers. It would seem reasonable to consider that the vertical canals fibers may be less resistant; the horizontal canal mechanism is the more active, the more frequently used, and unquestionably the more primitive. It would seem that the horizontal canal mechanism might be more resistant, because of its being more firmly established in function than that of the vertical canals.

For:

1. The conception of separate neuraxial pathways from the different canals was advanced in 1916, on the following data:

In a series of cases with intracranial lesions, in which the labyrinth and VIII Nerves were normal, the vertical canals failed to produce responses.

These cases included lesions of the pons, angle tumors pressing against the pons, cerebellar lesions causing pressure against the pons, absence of the IV Ventricle and internal hydrocephalus with pressure within the IV Ventricle.

These cases suggested the following:

If the lesions involving the pons caused a block in the responses from the vertical canals without impairing the responses from the horizontal canal, and as in all these cases the medulla oblongata was uninvolved, it was considered probable that the fibers from the vertical canals ascended into the pons, probably close to the floor of the IV Ventricle, whereas the fibers from the horizontal canal presumably traversed the medulla oblongata.

Confirming this viewpoint were the findings in the case of thrombosis of the posterior inferior cerebellar artery; all responses from all canals were normal, except that the right horizontal canal failed to produce vertigo and past-pointing.

This lesion, thus involving a portion of the horizontal

canal pathways, is known to involve the right side of the medulla oblongata in the region of the right inferior cerebellar peduncle.

2. Much confirmatory evidence of the same character as the above has accumulated. Eagleton had previously noted that in cases of angle tumors, the caloric test from the opposite ear gave negative responses; later, Eagleton and others, observed that in such cases, when the head was tilted backwards sixty degrees stimulating the horizontal canal, the responses came through.

It is now a common observation that there is a non-response of the vertical canals in lesions of the posterior fossa involving the brain-stem by infiltration or pressure. Few cases, however, have been seen in which the horizontal canal showed no response, while the vertical canals show normal responses.

3. We have recently seen two cases, which, however, have not been confirmed by necropsy, in each of which the left horizontal canal failed to produce responses, whereas the responses from all of the other semicircular canals were normal.

4. Although no histologic demonstration has been made of separate pathways from the different nerve receptors in the end-organ, yet there is evidence that the vestibular nerve fibers spread out to be distributed to the vestibular nuclei. The fibers from the vestibular portion of the VIII Nerve are for the most part interrupted in these nuclei, within a short distance from their entry into the brain-stem. They enter the brain-stem as a compact bundle, but their nuclear connections require a physical separation of all the fibers, preliminary to their termination. Complete ultimate physical dissociation of all these fibers must be accomplished, since each fiber has its definite destination in the vestibular nuclei, different from all others. These nuclei cover a large area; they involve the lower portions of the pons and the upper portion of the medulla oblongata and

extend from the lateral aspect of the brain-stem well over toward the median line. Without speculation as to the ultimate course or destination of the neurons from the various portions of these extensive vestibular nuclei, the physical separation of cell-groups in these nuclei—regarded as representing end-organ units—would in itself constitute a neuraxial differentiation of sufficient degree to answer the requirements of the concept given in the question.

Resumé:

While much remains to be learned of the intramedullary pathways and connections of the vestibular fibers, the evidence indicates that there is an appreciable separation of the neuraxial pathways of the fibers in relation to the horizontal canal and of the fibers in relation to the vertical canals.

V. Summary

Theoretical:

1. The available evidence suggests that the cortical area for the reception of impulses from the vestibular end-organ, is located in the temporal lobe in each hemisphere.

2. Different cell-groups within the vestibular nuclei represent the different physical units of the end-organ.

It is Probable:

1. That the otoliths and the cristae of the semicircular canals cooperate in function.

2. That the vestibular fibers are not distributed to the peripheral motor neurons, but to the neurons of the ocular coordinating mechanism.

3. That the quick component of vestibular nystagmus can be produced by a lower mechanism, without impulses from the cerebrum.

4. That the impulses from the internal ear, producing a sense of movement, pass through the cerebellum enroute to the cerebrum.

Reasonably Proven: *To be relied on in clinical work.*

1. There is a cerebral cortical center for hearing.

2. This center is reasonably proven to be in the temporal lobe. Also that each ear is in relation to the temporal lobe of each hemisphere.

3. Capillarity does not prevent endolymph movement within the semicircular canals.

4. There is a coordinating mechanism for ocular movement within the brain-stem.

5. Spontaneous vertical nystagmus, in patients who have other symptoms of intracranial lesion, indicates that the lesion is located within or adjacent to the brain-stem.

6. Perverted nystagmus after ear stimulation is indicative of involvement of the brain-stem.

7. Motion-sensing impulses from the end-organ, are conveyed to the cerebral cortex.

8. Tumors of the cerebello-pontile angle frequently show a typical neuro-otologic phenomenon-complex.

9. There is an appreciable separation of the neuraxial pathways of the fibers in relation to the horizontal canal and of the fibers in relation to the vertical canals.

Discussion.

Dr. Ernest Sachs, St. Louis, Mo. When the Barany tests were first described, I welcomed them as a new method of precision to help us in localizing cerebral lesions. As is the case with almost every new method, sober second thought has modified what was at first claimed for them. In fact, there was a period from which we are now emerging during which the pendulum swung the other way and now it has become somewhat stabilized.

These tests have interested me from two points of view, first, their value in helping us to localize a lesion with greater accuracy, and secondly, from a physiological standpoint.

I have had Barany tests done on forty-one cases of suspected brain tumors. All these tests were made by Dr. H. W. Lyman. I can only speak of the value of these tests to me personally. I find I must differentiate between what is of real value in locating a lesion and what is purely of scientific interest. In one of these forty-one cases the Barany tests gave information pointing to an intracranial lesion which was not suggested by any other clinical signs but that patient was lost sight of so that we had no confirmation on that case. There have been several cases in which the Barany tests were normal and in which no tumor was revealed at operation. At least one of these died but we were unable to get an autopsy to check up our findings. In every case of tumor of the posterior fossa confirmed by operation or autopsy the Barany tests have shown abnormalities. They have, however, led to confusion due to the fact that increased intracranial pressure may cause pressure on the pons and medulla thus interfering with the nervous mechanism by which these phenomena are carried out so that certain normal vestibular reactions are absent. It is, however, I believe practically impossible to

determine whether the involvement of the pons and medulla is due to pressure or due to involvement by a tumor. Dr. Lyman has made the point that bilateral absence of pontine reactions is much more likely to occur with internal hydrocephalus than unilateral and I think this is a very valuable observation. But I would like to point out that pressure symptoms may arise from an internal hydrocephalus or from pressure by a tumor and yet in neither case does it mean actual involvement by the growth. To the neurological surgeon who has to treat these cases, this point is of vital importance. In the hope of throwing more light on the subject I carried out an experimental study with Dr. B. Y. Alvis. As happens in so many researches, some of the points one hopes to throw light on and is most anxious to determine are not cleared up, and others which one did not think of come to light.

The vestibular nerve ends in three nuclei, Nucleus of Deiters, Von Bechterew and Nucleus Magno-cellularis. I believed and still do that since there were three semicircular canals and three nuclei, probably each nucleus controlled one canal. We tried to get evidence on this point but failed. We did, however, show that no fibres pass from any vestibular nuclei to the lateral lobes of the cerebellum. We also were unable to find any fibres passing from the vestibular nuclei to the nuclei controlling the eye muscles.

The vestibular fibres lie so close to the floor of the fourth ventricle and around the aqueduct of Sylvius that any dilation of the aqueduct would cause pressure on these fibres. The Barany tests are most frequently abnormal in lesions of the posterior fossa. Tumors of the posterior fossa in the vast majority of cases, fully 90% of the cases and probably even more have an associated internal hydrocephalus. It is this internal hydrocephalus which is of the obstructive type which is responsible for the disturbed Barany tests and this is the reason in my opinion why so frequently we get the report involvement of the pons.

If these facts are correct and I believe they are, the questions of greatest interest are these:

1st. Of how much value are the Barany tests for purposes of accurate localization?

2nd. Do the Barany tests enable us to recognize a lesion before there are other evidences of an intracranial tumor?

3rd. What place in our diagnostic work shall we accord the Barany tests?

My own answers to these questions are briefly as follows:

The Barany tests enable us to establish that a lesion is in the posterior fossa. These tests may indicate if a lesion is solely in the cerebellum but finding involvement of the pons does not necessarily indicate that the lesion has actually involved the pons since pontine involvement may be produced by an internal hydrocephalus or by increased intracranial pressure.

I have seen but one case in which the diagnosis of a posterior fossa lesion was made when all other neurological symptoms were absent and this case was never confirmed. It may be if we saw more very early cases, the Barany tests would show abnormalities before any other neurological signs are recognizable. I understand that Dr. Fischer in Philadelphia has had a number of such cases. Based on my own experience it seems to me that the Barany tests are of value as confirming our diagnosis based on other findings but they do not enable us to locate lesions with great accuracy. The one exception to this statement may be in the case of tumors located in the cerebello-pontine angle for which Drs. Jones and Fischer have described a very characteristic symptom complex.

Particular care must be exercised in interpreting the significance of pontine involvement. I doubt whether we are ever justified in deciding that a given lesion is inoperable because these tests indicate that the pons is involved.

Professor H. C. Bazett, Philadelphia, Pa.: I am very appreciative of the honor of being asked to discuss this presentation. My point of view is based on the more

scientific aspect of the question, but I think the scientists and clinicians have common objects. Dr. Sachs's remarks were very interesting. That internal hydrocephalus causes blocking of these tracts is not only of clinical importance but of scientific interest, so that the fields of the scientist and clinician overlap to a large extent.

I would like to congratulate the readers of the paper on their well balanced outlook in comparing clinical evidence with experimental work. I agree that it is difficult to get any experimental evidence with small discreet lesions, but we can make big lesions in various positions and see how the symptoms of the animals differ afterwards. Perhaps the clinician is liable to criticism often for not reporting pathological conditions in sufficient detail; it must be recognized that a minute difference in the position of a lesion may make a great contrast in the physiological reaction. I have been working with decerebrate cats in which everything has been removed above the tentorium. There is often a reflex response to sounds; wagging of the tail is seen and often after the third day they lift their head to one side in reacting to sounds. It is of some importance for the neurologist and otologist to recognize that, in animals at least, movements may be made in reactions to sounds quite apart from consciousness. But this reaction which is seen if the section passes from the anterior margin of the superior colliculi to emerge ventrally two mm. in front of the pons is not seen if the section passes two mm. posterior to this on the ventral side (i. e. to the anterior border of the pons): similarly the reaction is seen if the section goes from the middle of the posterior colliculi to two mm. in front of the pons, but is not seen if it passes from the posterior edge of the inferior colliculi to two mm. in front of the pons. Consequently, it is a question of millimeters but it is possible to localize the upper limit anatomically if the reflexes are concerned. By an exact recording of different lesions and the accompanying symptoms it should be therefore possible to arrive at exact localization. But if the clinician may be sometimes

criticised for a neglect to make a sufficiently accurate autopsy, the experimental workers are generally liable to much severer criticism in that they often examine their animals from one standpoint only, and in particular they have usually neglected to make vestibular tests.

Turning to the more definite problem of Physiology in relation to the auditory tracts, it is difficult to destroy experimentally all the auditory paths, showing a strong similarity with clinical findings. A description has been recently given by de Barenne[1] of a cat in which the whole cerebrum had been removed except the archipallium (the olfactory area), though there was possibly a minute amount of the auditory area left. The internal geniculate bodies and the geniculate temporal tracts which are supposed to carry all auditory sensations to the cerebrum were found on autopsy to be completely degenerated, and yet the animal while alive was not only able to hear but was able to localize sounds and would go towards anyone who whistled. The possibility of other tracts being in existance besides those usually described must therefore be considered.

In regard to the labyrinth nystagmus tests have been rather neglected by the experimental worker. Magnus [2] has thrown a certain amount of criticism on the use of nystagmus after turning as a test of labyrinthine function on the ground that the mechanisms involved extend over a large portion of the brain. The centers involved must obviously extend at least from the nucleus of the third nerve to the nucleus of the eighth nerve. In consequence, he has tried other methods of approach, utilizing his position reflexes and also the effect of turning on movements of the neck, since animals often show a jerky movement of the whole head and neck similar to the eye nystagmus and in the same direction. He found in rabbits that if the cerebrum was removed and the brain stem cut below the pons but in front of the trapezoid body you can get a

(1) Barenne de-Arch. Néerland. de Physiol. 1919, 4, p. 31.
(2) Magnus-Arch. fur die ges. Physiol. 1916, 163, 405.
de Kleign and Magnus-Münch. med. Woch. 1919, 20, 523.

normal type of neck nystagmus on turning. This is true also even after the removal of the cerebellum. He also considered that in rabbits perfectly normal eye nystagmus (for these animals) could be attained when the cerebrum and thalamus had been removed but where the mid-brain had been left intact. Consequently, he considered that the nystagmus depends on the interraction of stimuli from the eighth nerve and its centers with others from muscles of the neck or the eye and that these are the only factors required. This disappearance of the nystagmus with sections in the region of the pons or higher he considers to be caused by the cutting off of the connections with the eye nuclei. While nystagmus may then be regarded as an alternation in balance between labyrinthine sensations and muscular sensations and may be compared, as has been done, with a reflex such as the scratch reflex it must be remembered as Dr. Wilson has pointed out to me, that all nystagmus is not necessarily of the same character. Scratch reflexes for instance are well seen in a spinal animal, are much more difficult to obtain in a decerebrate preparation, but if obtained much more complicated reactions may be seen in the decerebrate than in the spinal animal.

The absence of degeneration extending to the cranial nerve nuclei after section of the eighth nerve is not surprising. It is very probable that several neurones may be concerned in such reflexes. It should be noticed for instance that Simpson[3] has described degeneration of the pyramidal tracts as extending not into the anterior horns but into the posterior in the spinal cord, while in the brain stem he found the fibres ended not in the eye muscle nuclei but among the cells of the formatio reticularis.

One other point I should like to make. I do not think that the sensory center for the labyrinth in the cerebrum will ever be found. It is impossible to think of any sensation which is not bound up with labyrinthine sensations. The cerebrum seems to deal with concepts not sensations, so that the occipital lobe, the Rolandic area,

(3) Simpson-Proc. Scott. Micr. Soc. 1902, 3, p. 158.

etc. deal not with simple visual and muscle sensation, but with visual and muscle concepts in which these sensations are combined with sensations of position, many of them labyrinthine.

We have made our students recently do Barany chair tests and have questioned them carefully about their sensations. When stopped keeping their eyes shut they feel that they are turning in the opposite direction to their previous movement, but if they open their eyes the sensation is reversed and they feel that they are going around in the same direction as their previous movement. Similarly if a student is turned leaning forward on opening his eyes he seems to be turning in the same way as he was previously turned if he keeps his head in this position, but if he raises his head to the vertical position, the sensations are no longer interpreted as turning but as an abnormality of the sense of the vertical, while if he puts his head right back and looks at the ceiling he now appears to be moving in the opposite direction to his previous turning. With the eyes open the usual sensation is that the room is turning in the direction of the quick component of nystagmus. These well known sensations surely indicate that the cerebrum is dealing not with visual sensations but with concepts combined from retinal, labyrinthine and muscular sensations. so that it would seem as though the various tracts for visual sensation, conscious muscle sensation and skin sensations all have a relay in the thalamus in order that they may be recombined and correlated together, and merged also with labyrinthine impressions, so that a sensation of position is included in the cerebral conception. Such an analysis of these sensations therefore makes it very improbable that there is any special area of the cerebrum devoted particularly to labyrinthine sensations.

Dr. Harris H. Vail, Cincinnati, Ohio: Obviously it is possible to discuss only a small portion of this most interesting and scientific work on certain neuro-otological problems.

Dr. Weisenburg, in a recent article[1] says "that perhaps because of the war otologists have learned to apply the Barany tests, but few neurologists have practical knowledge of the tests or of their applications in neurological diagnosis, consequently otologists have been led to furnish diagnoses of intracranial lesions, obviously exercising a function outside of their sphere. Clearly these tests should be performed by neurologists with otological training or otologists with neurological training."

Apparently the popular conception of neuro-otology is that otologists by performing the so-called Barany rotation and caloric tests are able to make diagnoses of intracranial lesions by these tests alone, where as a matter of fact we all deem it is impossible to make any short cuts in the localization of intracranial lesions and otologists working in that field must avail themselves of all the neurological examinations. Dr. Weisenburg apparently sounds a very apt warning and we all should heed it.

My own feeling is that we should not attempt to make a diagnosis of intracranial lesion by the rotation and caloric tests alone. We should go through all the examinations that the neurologist or neurological surgeon makes and then examine our patient by the rotation and caloric tests which Dr. Jones has put on such a standard basis. If these latter tests conflict with the clinical picture the best thing for the examiner to do is to be a bit cautious and conservative about making a definite diagnosis of intracranial lesion, repeating the tests until the findings are constant and can be checked up by any other observer. Doing these tests without anyone to check up our findings is apt to make us perhaps a little prejudiced in our interpretations. As, for example, in a case where we suspect the presence of an eighth nerve tumor, knowing that we should not obtain reactions on the side of the lesion, in such a case we are apt to disregard any ocular movements observed during the caloric test and report no nystagmus;

1 Archives of Neurology and Psychiatry, Vol. 7, No. 2, February, 1922, page 213.

whereas in the same case we feel certain from our clinical examination that the lesion is not an eighth nerve tumor but say a cerebellar tumor, we shall give the same patient credit for these vague ocular movements and report a slight response from this particular set of semicircular canals. To my mind this is one of the dangers of the rotation and caloric tests, but it is absolutely essential that we do make these complete examinations before doing the rotation and caloric tests as I think we shall thereby perform our tests better and get a smaller percentage of error between our findings by the tests and the actual pathological findings in the case. Sometimes we are pleased at the reliability of these rotation and caloric tests on patients with intracranial lesions.

I recall a good example of their worth in a case of Dr. Harvey Cushing at the Peter Bent Brigham Hospital. In brief the history is as follows:

F. N. aged forty-one, with all the symptoms of a left-sided cerebello pontine angle lesion except no evidence of increased intracranial pressure.

On examination there was found complete left facial and abducens paralysis and anesthesia of the left cornea, together with a brisk spontaneous nystagmus to the right and left with a rapid vertical nystagmus on looking up. Fundi showed no evidence of any choking or hyperemia.

There was marked falling in Romberg's position and marked incoordination of legs and some of hands. Knee jerks were active with Babinski on the left side. Spontaneous past pointing by the left arm was present with coarse tremor. Hearing: left ear moderate loud conversation only was heard. No forks heard and upper limit was 2.5 on the Galton whistle. Right ear normal hearing with Weber referred to the right. Both membrana tympani normal.

Rotation and caloric tests were done on the second day after admission and absolutely no after-nystagmus could be produced. After-vertigo on rotation tests was practically nil, certainly not more than two seconds and no past pointing was obtained.

At this time I presented a diagnosis of cerebellar pontine sclerosis, bilateral and worse on the left side.

An examination ten days later showed no after-nystagmus on right and left rotation with head 30° forwards. No vertigo or past pointing. On the caloric test no reactions present from douching the left ear, head forwards or backwards. On douching the right ear with the head 30° forwards, no nystagmus was obtained after five minutes irrigation. On putting the head back there was a slow slight indefinite horizontal nystagmus to the left.

My conclusions at this time were that the reactions were possible from left acoustic neuroma but owing to the lack of increased intracranial pressure signs they would suggest a diffuse lesion through pons and cerebellum, bilateral but worse on the left side.

Three weeks after admission Dr. Cushing performed a sub-occipital exploration with exposure of a small tumor mass projecting into porus on left side. Partial enucleation of nodule. The tumor was proved to be a glioma involving the pons.

Another very interesting case of Dr. Harvey Cushing is as follows:

L. S. aged eleven, with the history that at four years of age he fell out of baby buggy and was unconscious for three days. Had a vomiting period lasting some months with dizziness and headaches. Ten weeks before admission an acute attack of tonsillitis with recovery after three weeks. Five weeks before admission he vomited before breakfast with slight nausea and frontal headache. Five days before admission a left internal strabismus was noted.

A history was obtained of dizziness, vertigo, nausea, cervical rigidity and sub-occipital tenderness. Slight ataxia was found subjectively more on the left. Positive Romberg with staggering gait and falling to right or left. The most marked feature of this case was the vomiting. The hearing was normal and fundi showed 3.0 diopters swelling.

Examination showed spontaneous nystagmus horizontally to right and slightly rotary to left. The weakness of both

external recti muscles was not synchronous, i. e., there was some paralysis of associated lateral movements such as Freeman[2] has lately described.

Spontaneous past pointing was present on the right side with incoordination, hypermetria and awkwardness. Deviation usually to the left. Left arm touched properly. After right rotation, a good horizontal after-nystagmus to the left for 24 seconds was obtained. After-vertigo was 21 seconds. Past pointing was not tested. On rotation to the left both eyes were drawn to the left then slowly rolled to the right and back to the left with a few rapid fine horizontal movements to the right. This reaction was quite suggestive of an incomplete conjugate deviation. Duration was 26 seconds, after vertigo 10 seconds. Past pointing not tested.

With the caloric test of the right ear, with the head 30° forwards, a fair rotary nystagmus to the left was obtained after 1-½ minutes. There was present however a conjugate deviation of both eyes to the right, with a rare rapid jerk to the left. Past pointing not tested.

Caloric test of the left ear with the head 30° forwards, gave no nystagmus after five minutes. With the head 60° backwards, a brisk horizontal nystagmus with quick component to the right ensued.

At this time my impression was a lesion of the right cerebellar hemisphere involving the right superior cerebellar peduncle. A few days later Dr. Cushing performed the cerebellar operation on this case and found a right-sided cystic glioma of the cerebellum extending down through the foramen magnum, overlying the medulla. A large cyst containing 60 to 75 c. c. of straw colored fluid was found in the right cerebellar hemisphere. A mass the size of a pigeon's egg was enucleated along with cyst wall. There was left a large cavity in the cerebellar hemisphere. The tumor had extended forwards considerably.

There was in this case a tendency for conjugate deviation from stimulation of the right semicircular canals. In this

2 Archiv. of Neurol. and Psych., Vol. 7, No. 4, April, 1922.

case with an enormous tumor and marked pressure in the posterior fossa it was possible to obtain reactions from the right semicircular canals and also from the left horizontal semicircular canal: the vertical canals on the left side apparently being blocked by the pressure. Just why this should have occurred I cannot explain except that the pressure somehow or other did not involve the vestibular fibers on the right side. The presence of conjugate deviation of the eyes after vestibular stimulation is not pathognomonic of supratentorial lesions, and I quite agree with Drs. Ingham and Jones on the point they make about the presence of conjugate deviation of the eyes in lesions of the posterior fossa.

In regard to the cerebello-pontile angle tumor phenomenon-complex, there are undoubtedly variations in the responses from the vestibular apparatus on the side opposite to the side of the lesion, varying from normal responses to absent responses from vertical canal stimulation such as Drs. Ingham and Jones state, and furthermore it is quite possible to have variations in the tests repeated within short intervals of each other, that is to say this phenomenon of absence responses from the vertical canals on the side opposite to the lesion is very likely due to pressure in the posterior cranial fossa and we know that this pressure varies from time to time. I feel that there is no definite syndrome in acoustic neuromas. The auditory function is not always entirely destroyed on the side of the lesion. One other fact well brought out by Drs. Ingham and Jones is that in cerebellar lesions the after-vertigo is decreased. This fact should be considered in all past pointing tests where we find decreased vertigo and it is important in doing our past pointing tests after rotation to test the arms in different order, as for instance, on right rotation, test the right arm first then the left arm: repeat the right rotation testing the left arm first. Personally, I am beginning to disregard the past pointing tests more and more although I do them routinely I attach very slight

significance to abnormal reactions unless they fit in with the rest of the symptom complex.

One other case of interest is that of O. S. a patient of Dr. Cushing at the Peter Bent Brigham Hospital. Dr. Cushing has reported this case in detail in the Laryngoscope, August 1921.

It was a case of right acoustic neuroma with a history of probably twenty years duration. Four distinct examinations by the rotation and caloric tests were made on this patient and in all of them there was found on left rotation a tendency for conjugate deviation of the eyes to the left. Hearing was normal in the left ear with marked deafness in the right ear, loud shout only being perceived. Caloric tests on all four occasions gave absolutely no response from the right vertical canals. On two occasions a very slow diminished response from the right horizontal canal was present and on three occasions there were present diminished and dull responses from the left vertical canals with normal responses from the left horizontal canal on all tests.

This patient was operated on by Dr. Cushing with an exposure of a large right-sided acoustic neuroma.

Passing on to another subject of extreme interest and importance to the otologist, I should like to discuss briefly the rotation and caloric tests in cases of chronic suppurative otitis media with labyrinthine symptoms.

The following case, J. B. aged twenty-eight was on the service of Dr. Philip Hammond at the Massachusetts Charitable Eye and Ear Infirmary. In brief: He had a left chronic suppurative ear for sometime and in the week just before admission to the hospital became very dizzy with some vomiting.

On examination there was a spontaneous horizontal nystagmus to the right and left fairly well sustained of fair amplitude and greater on looking to the right.

Hearing in the right ear normal. In the left ear conversation at one inch. Lower limit 1024 d. v. Upper limit 0.8 with Galton whistle. Weber referred to the left. Condi-

tion of the ear a foul, suppurative one with loss of ear drum membrane. Wassermann negative. Neurological examination normal except for markedly contracted pupils.

No spontaneous past pointing was present. On rotation to the right there were a few rotary jerks and the fine spontaneous nystagmus but no true after-nystagmus. After-vertigo was 3 seconds.

Past pointing tests showed 2 inch deviation to the right with both arms, testing them in different order.

Rotation to the left: a definite horizontal nystagmus to the right of 18 seconds duration. After-vertigo 7 seconds.

With the fistula test: On compression in the left ear a violent horizontal nystagmus to right with vertigo and past pointing to the right with both arms for about one foot ensued. On suction a less violent but very definite horizontal nystagmus to left with less vertigo. Past pointing reactions were not tested.

On first introducing the cold water into the left ear there were some rapid horizontal jerks to the right similar to those induced by the fistula test. No rotary nystagmus appeared until after 2-½ minutes of irrigation. Amplitude large.

Head 60° back: There was no after-nystagmus and no past pointing.

On douching the right ear, there appeared a rotary nystagmus to the left of fair amplitude and speed. Past pointing was to the right.

The impression at that time was that there was a fistula in the left horizontal semicircular canal with circumscribed labyrinthitis, and a lesion involving the fibers from right labyrinth to posterior longitudinal bundle fibers. No evidence of cerebellar lesion. A few days later Dr. Hammond performed a left radical mastoid. The fistula was found in horizontal canal eminence. One week later skin graft.

Patient complained of no vertigo and was discharged three weeks later. At this time on rotation to the right

there was no after-nystagmus. On rotation to the left brisk horizontal after-nystagmus for 15 seconds.

Fistula test negative in the left ear.

Two weeks later was examined in the out-patient department: No vertigo or headaches. Left ear discharging a bit. Was at work feeling well.

Examination showed hearing in left ear absolutely nil. No bone conduction present. On rotation to the right: No after-nystagmus, vertigo or past pointing. On rotation to the left: A fine horizontal after-nystagmus to the right duration 18 seconds. After-vertigo 11 seconds. Past pointing: Left 8 inches to the left; right 6 inches to the left.

Caloric test of the left ear, water 65° F. Head 30° forwards.

No after-nystagmus after 5 minutes was obtained.

Head 60° back: No nystagmus.

Fistula test negative.

In other words in this case the rotation and caloric tests have shown us the progress of the condition from one of circumscribed labyrinthitis to a diffuse labyrinthitis with destruction of the labyrinth on that side and for this reason I have reported this last case as it affords a striking example of the value of these rotation and caloric tests in the field of chronic suppurative otitis media.

Dr. Wells P. Eagleton, Newark: My interest in the vestibular symptoms is entirely that of a clinician, but performing a large number of operations has led me somewhat away from the clinical aspect and given me a certain philosophy in regard to the ear. There has been great confusion on this subject. I think the mistake has been made of treating the vestibular apparatus as of a very high order of nervous function. We have been talking about the vestibular tracts the same as we have about the speech tracts, and they are entirely different biologically. A man receives a blow over the left temporofrontal region and becomes speechless. Speech is the highest form of nervous

development we have outside of the higher psychic functions which grow out of it. Hearing is only present in vertebrates. Speech is a recent addition to life. When we go below hearing we come to some of the fundamental phenomena of life itself. There is no evidence of hearing much below the vertebrates, and the vestibular apparatus is well developed in the invertebrates and in the very low forms of life. This is a biological fact that we are dealing with.

Professor Bazett has called attention to the fact that the whole upper portion of the nervous system in animals has been removed, yet there has been nystagmus. What does this mean? It means that in certain parts of the vestibular apparatus, all function helps with orientation. Dr. Jones has shown this by pictures and that is the only way we shall ever learn. If two men see the same thing, they do not see it in the same way. There is the personal equation, so that it is only by a permanent pictorial record that we shall ever get anywhere. I congratulate Dr. Jones on what he has shown. He removes a large part of the cerebellum. After a time the cat falls well,—not perfectly, but still fairly well, because compensation has immediately commenced in the lower forms of nervous tissue. In the very low forms of animal life you can cut the creature in two and it will still live. Dr. Tilney has worked out a philosophy about the cerebellum which makes it understandable. In studying the dog fish he has worked out his conclusions. The cerebellum first appears in the fishes; it is not in the invertebrates, but there is a vestibular apparatus in the invertebrates. The fundamental cerebellum is the bulbar and is part of the primary spinal cord and in that are found the vestibular nuclei. Later there is added a posterior and a medial lobe which we regard as the vermis and later the two hemispheres. These are only present in animals which acquire use of the four extremities. In the cerebellum the lowest part represents the vestibule; the middle part the orientation of the trunk; the third part movements of the four extremities. Accepting

this as true, what is clinically evident? Gray took a cerebellum and with a knife cut below the cortex and above the grey matter, and produced certain movements or disturbance of orientation. It disappeared in from 4 days to 2 weeks. The cerebellum itself has to do with movements in general and has a very low form of nervous development, easily compensated for. How can we apply this fact? We can apply it this way, that we must not look for a localization from the vestibular tests. It does not exist. Minute lesions such as small hemorrhages do produce syringomyelia and these are shown by isolated manifestations of the vestibular apparatus which makes the diagnosis of lesions possible. But we are dealing with gross lesions of the nervous system. Localization of the vestibular apparatus is very definite, but the localization of the intracranial portion is, in the majority of cases, impossible by the vestibular tests alone.

There are certain factors which stand out prominently: one is evidence of increased intracranial pressure as shown by the vestibular tests. I reported 3 cases of tumor of the brain with aural manifestations. I syringed cold water in the opposite ear. There was no nystagmus. I did not know that if he turned the head upwards he had no nystagmus. The reaction of the vertical canal on the opposite side confirmed this as evidence of intracranial pressure. I made the assertion that in regard to importance from the surgical standpoint this precedes ocular manifestations, that is the papilledema which has been our sheet anchor. That is if the vertical canals did not react to the cold caloric in the upright postition there was increased intracranial pressure of the posterior fossa, not necessarily of the cerebrum. That has not been established in a great many cases. I have come today to regard many of the so-called defences of peripheral as of nerve origin, or intracranial origin. We do lumbar puncture and we will find the fluid under pressure and the chemical composition of the fluid very often altered.

I have also come to regard as of great importance the diminution of the duration of induced nystagmus by turning. I am sure that we must separate induced nystagmus by caloric from the nystagmus by turning. One is the thermic stimulus and one is mechanical. Dr. Jones showed a man touched with a probe, and this caused nystagmus. Turning him did not cause it; the caloric did not cause it. Different forms of nervous stimuli should be separated. If you turn a normal man he has nystagmus; if you keep on turning him it stops. It cannot be induced after a certain time. In intracranial lesions of the posterior fossa, in studying fractures of the skull, I found this: if a person receives injury to the posterior fossa and the spaces of the pia-arachnoid are filled with blood, you get bloody cerebrospinal fluid. Turning that man gives nystagmus reduced to fifty per cent, yet his hearing is perfect. I discovered this in studying effects of induction of protective meningitis, by my labyrinthine operations. I induced a protective meningitis. There were many cells and cloudy fluid. The duration of nystagmus was reduced 50 per cent. In fractured skull cases this rule applies and it lasts a long time. In the chair the patient had reduction of nystagmus on turning, but the caloric was good. I have postulated 2 things: one the absence of reaction of the semicircular canals to the cold caloric means intracranial pressure; two, the reduction of the nystagmus on turning masses filling of the cerebrospinal spaces of the posterior fossa by either exudate or hemorrhage.

Dr. J. Gordon Wilson, Chicago: We all agree with Dr. Jones that moving pictures such as he has shown, are an excellent method to study and record pathological lesions and clinical states. It is impossible to discuss adequately Dr. Jones's most interesting paper, in which he summarized clearly and states briefly, the "for" and "against" of many of the debatable points in vestibular physiology and pathology. I will confine my remarks to two points in the many controversial issues which his paper raises.

(1) I am still unconvinced that the cerebral mechanism above the corpora quadrigemina has no influence on the quick movement of nystagmus. Dr. Jones has well enumerated the arguments against this central origin of the quick phase. Exception might well be taken to each of the arguments so advanced. I will refer only to two. There are authentic clinical observations which point to a central mechanism anterior to the corpora quadrigemina being active in man and the higher mammals as outlined in the paper I published with Dr. Pike, 12 years ago. Evidence that lower centres, presumably in the pons, are the seat of origin in man, will require experimental evidence in animals whose nervous system is higher than the rabbit, in which animal most experimental work has been done. Further, in arguing against the cerebral origin in higher mammals, such as the cat to which Dr. Jones refers, we will require evidence that necessitates no assistance from the theory of surgical shock to explain its momentary duration or after stimulation.

In regard to (6) few physiologists will regard the scratch reflex as observed in decerebrated animals as comparable to the scratch reflex in the same animal before decerebration.

(2). I doubt whether we can call the vestibular sense a "specific special sense". The information derived from the vestibular mechanism in the ear is too unreliable to have it so designated. An end organ part whose function is to sense acceleration of movement and which gives information in regard to movement during acceleration which may be 50% wrong, can scarcely be regarded as a "special sense organ."

Dr. Tom Williams, Washington: I have not had the time to study the question sufficiently. I want to speak about Dr. Wilson's remarks. There is confusion on the whole subject of nystagmus. I do not think Drs. Jones and Ingham were speaking of a single phenomenon. Rapid movement of the eyes in one direction with slow return

is one thing. There are other movements of the eyes which are oscillatory. I think these are a different phenomena, distinct from true nystagmus. I think the last speaker's remarks are well taken. Movements provoked by experimental alteration of the conditions in the labyrinth are of the same order as conjugate deviation of the eyes, which can be determined purely through the lower neurone sensory's motor system of the vestibule. The movement of return to the normal axis is determined by something above the corpora quadrigemina, by factors that intricate with vision. The behavior is different with the eyes open from that when they are closed. With reference to the last problem, can we rely on the experimental observations or the clinical and pathological ones, or both? Can we be sure that there are not separate paths from the horizontal canals and the vertical canals respectively? I think we can. There are many clinical factors which point to that disassociation although we have not yet ascertained the exact path for the horizontal canal. For instance, in a woman with cerebellar symptoms we operated, believing we might possibly find a new growth. On the left side, the impulses from the vertical canals were retained, whereas all other impulses had disappeared. What we all found was a symmetrical lesion involving both posterior cerebellar arteries. This had permitted of variation in the vestibular distribution going to the semicircular canals on the one side. We must therefore assume this dissociation.

Dr. E. A. Crockett, Boston: After thirty years of the practice of Otology, I feel like a student. The papers this morning have been so unusual and so excellent, that I move a vote of appreciation by the Society be offered to those who have contributed to the success of this meeting.

Dr. Dench: I second the motion. (Carried).

Dr. B. Alex. Randall, Philadelphia: I have been asked to speak in this discussion of the paths of the cochleal

fibers, (of which Dr. Jones rightly says that we have of late learned little new) but prefer to do so mainly with pictures on the screen, since these are to most of us more explicit. We elders have had somewhat to relearn our anatomy ,and trace the afferent fibers of the sense-organs from their peripheral ganglion-cells to the apparent, and if possible their ultimate, "origins" in the central nervous system.

We know that involution of the epiblastic cells form on each side of the head the otic pit; that this cuts off as a vesicle separate from the surface, which then develops into the complexities of the membranous labyrinth. While this somewhat parallels the development of the cerebro-spinal axis, its cells are not therefore neural any more than those of the crystaline lens of the eye, identically formed: only those cells connected with the brain by dendritic fibers of the neurones of the spiral ganglion of the cochlea or the Scarpa ganglia for the vestibular endings become 'hair-cells' supported by their otherwise modified neighbors to form the Corti organ the ampullar crests. The neuraxial fibers, grouped as the VIII nerve in the internal meatus, pass to the brain-stem where the cochleal fibers enter the ventro-lateral or accessory nucleus, whence some of them ascend to the dorsal nucleus or acoustic tubercle external to the Restiform body, while others cross through the Trapesium to the Superior Olive of the same and the opposite side. The dorsal fibers appear as the acoustic striae in the floor of the Fourth Ventricle or beneath it to decuss in part, although some remain homolateral; and passing through the Lateral Fillet, relayed by its nucleus, reach the Geniculates and by the Acoustic radiations reach to the cortex of the First Temporal Convolution. Supplementing this course is a secondary system following slightly different paths; while a third set of fibers reverses, according to Held, these lines and links in downward order the nuclei of the other paths. Herein may lie the explanation of the rare occurrence of deafness in brain-stem

lesions; but it also shows that many fibers from both cochleae are within reach of a limited lesion below the floor of the Fourth Ventricle and could be simultaneously blocked, causing total deafness as in epidemic Meningitis.

One peripheral point has received little notice. We know that the cochlear nerve is made up of the neuraxial fibers from the spiral ganglion passing through a spiral tract in the bottom of the internal meatus, so that those from the apex form its center clothed by the layers of fibers coming from the successively lower turns. These later form the periphery of the nerve-bundle protecting those within: so we realize why the high tones, which have their neural paths from the lowest cochleal turn, are usually the first lost in lesions of the internal meatus or the pontine angle. The distribution of the optic fibers at the chiasm is of recognized diagnostic value in respect to pituitary growths; but little if any attention has been given to this anatomic relation—the greater vulnerability being rather ascribed to the nearness of the Corti end-organ for high tones to the membrane of the round window.

AN UNUSUAL CEREBRAL COMPLICATION OF MASTOID OPERATION, RELIEVED BY SUBDURAL DRAINAGE.

By E. A. CROCKETT, M. D., Boston, Mass.

In two papers, one read before the American Otological Society in 1908 and the other before the American Laryngological, Rhinological and Otological Society and published in their Transactions in 1916, Vol. 22, page 173, I have called attention to the possibility of relieving apparently hopeless cases of meningitis by the opening of the dura mater in the temporal region and the placing in of a drain for the free exit of cerebrospinal fluid.

This case is an extreme illustration of the benefits which may occasionally derive from such a surgical procedure.

Julia R. 33 years old was admitted to the Massachusetts Charitable Eye and Ear Infirmary on January 3, 1922. She had had a chronic suppurative discharge from the left middle ear for many years. During the last year her family had noticed a decided change in her disposition, otherwise she had had no ill results from the chronic discharging ear until 6 weeks before admission, when she began having severe pain in the left ear which had continued more or less all the time up to the period of admission. The discharge during this period of pain was less than usual, it was also noticed that she was dizzy upon standing and fell to the left.

The mastoid operation was performed on the left side by the Senior House Surgeon assisted by one of the Staff. During the course of this operation, the lateral sinus was exposed and it was observed that there was a sinus in the horizontal semi-circular canal.

About six hours after this operation the patient had a good deal of cerebral disturbance, not exactly delirium, but evidently an acute cerebral condition. She was given ⅛

gr. of morphine. At 8 P. M. the house surgeon noticed the patient was rational, but complained a good deal of headache.

At 11 P. M. the patient had projectile vomiting, also some nystagmus, but cerebrated perfectly.

At 3 P. M. she vomited and passed into a state of coma from which she could not be roused. There was marked disturbance of the pulse rate,—at times 78 and strong and at times 128 and intermittent, the eyes were strongly drawn to the left side, the pupils were dilated and in about an hour patient had a slight chill. Respirations were markedly irregular and of the Cheyne Stokes type. The eyes at times were deviated to the left, other times there was spontaneous, horizontal nystagmus to right. At times the patient was in complete coma, at times restless, at times incoherent attempts to talk and there were many convulsive movements of the muscles of the right lower leg and foot. When very restless, both sides of body moved freely, but when quiet there was almost no movement of the left arm, leg or left side of face.

The dressings and pressure wicks were taken out of the mastoid wound.

When I first saw her at 8:45 A. M. the following morning, about twenty hours after the mastoid operation, the patient gave a typical picture of the last stages of lepto-meningitis of the base of the brain. She was violently delirious, had to be restrained in bed, eyes wide open and staring, convulsive twitching movements and pounding of the right foot and arm; at times cried out violently and sat up and stared and cried "mamma;" both sides of face moved well and equally; triceps and knee jerks hyperactive; abdominal reflexes were absent; moves all parts of both sides of body; plantar reflex present; Babinski on left side; lungs and heart negative—abdomen negative; pupils contracted, but patient had had 1/3 gr. morphine in last 24 hours; fundi negative; slight vascular engorgement; apparently no photophobia. The patient had acute mania

and resisted all attempts to obtain neck sign; resisted bending head forward, but at times easily bends forward voluntarily; resists more with right than with left leg. The head showed a tendency to be turned to right; very restless from time to time and at these times the movements of body were not equal, the right foot pounds but never the left.

Blood presure 110/60, White count 30,000, Temp. 100, Pulse 60, Resp. 16.

A lumbar puncture was done and 10 cc of spinal fluid was removed under heavy pressure. The fluid was very thick and turbid; cell count 20,000. A smear from the spinal fluid showed numerous diplococci. A Gram Stain smear failed.

From observation of the patient's condition at this time, my diagnosis was that she had a rupture of an intracranial abscess, owing to the trauma necessary in doing a radical mastoid operation.

I found myself quite unable to make a differential diagnosis between a temporosphenoidal and a cerebellar abscess, but in view of the numerous convulsive movements to the right arm and leg, it seemed to me to be more likely temporosphenoidal. The patient's condition seemed to be quite desperate and any chance of recovery from operation absolutely hopeless, but it seemed to me well to make an attempt, but I personally thought she would die on the operating table.

The left side of the head was shaved on the operating table and a horseshoe shaped incision was made above the left ear, base upwards and the skull was opened just over the external auditory meatus with a ¼ inch trephine and rapidly enlarged with a rongeur. The exposed dura looked normal and there was no increase of intracerebral pressure. The dura was opened over the temporosphenoidal lobe and there was no attempt of herniation of the brain substance. The temporosphenoidal region was explored thoroughly with a brain knife and a trocar inserted five times in search

of an abscess, but none was found. One of these probings produced a bloody flow of cerebrospinal fluid evidently from the lateral ventricle, but no abscess was found. There being no evidence of rupture of a temporosphenoidal abscess, I then considered it possible that the patient's condition might be a meningitis and consequently followed the procedure indicated in the two papers mentioned above and a piece of rubber dam was carried between the dura and pia over the petrous portion of the temporal bone about as far as the internal auditory meatus, a stitch was taken through the drain to the edge of the dura, the external flap was sewed up with interrupted sutures and a dry dressing applied. The patient was taken from the operating room in good condition and placed in a special bed surrounded by padded boards.

For the next twenty four hours, she had almost incessant convulsive movements of the right side with only a few quiet moments between, so much so that the patient's arms and legs showed numerous bruises.

On my visit the following morning, the patient was absolutely unconscious, involutary evacuation of urine, eyes staring and deviated to left with occasional nystagmus to right, marked neck retraction and Kernig both sides, mouth wide open, head drawn to the right, seldom moved the right side of body, frequently moved the left, blood pressure 120/80, white count 22,000, was unable to swallow and had been given some food by rectum.

Forty eight hours after operation patient had retention of urine, which had to be drawn by catheter, had passed a restless night, but was now very much quieter. She had attacks of convulsive movements every few mnutes, but less violent in character. She seemed at times to recognize those around; head less retracted, mouth still kept open; Kernig still positive. There was a very marked discharge of cerebrospinal fluid necessitating the changing of the bandage twice a day.

Three days after the operation the patient was able to swallow freely and was given egg and milk by mouth and she seemed to recognize those around her for short periods. She had slight mania at times and marked aphasia and disorientation.

Five days after the operation, the patient had passed a good night, fairly rational most of the time and slept for periods of three to three and a half hours, took milk and egg nog, orange juice and cocoa, but still had periods of restlessness, but was less difficult to manage. There was a continued free discharge of cerebral fluid.

Six days after operation, the patient recognized her brother and said, "Hello Fred, where's Mother?"

The house record states, temp. 98. Resp. 20, Pulse 96. Eyes rest in mid position—head still kept back and lies on right side—has taken adequate nourishment by mouth.

Seventh day, white blood count 12,000.

Tenth day, white blood count 9,500.

Kernig and head signs have disappeared. Patient cerebrated perfectly well. Sat up in bed for short periods.

On this day a second lumbar puncture was done showing a clear fluid with cell count of 11.

Two days later the patient's white blood count was 8,000 and in view of her excellent condition, I considered it wise to take out the drain; up to this time there had been a profuse continued discharge of cerebrospinal fluid from the wound. From this time on the patient made an absolute recovery.

On the twentieth day after the operation, the patient was up out of bed and seven days later the record states she had been up all day for several days.

One month after the operation, she was discharged to the out-patient department.

On the 19th of March, the patient returned to the hospital for observation. The neurological examination was negative. The patient had no nystagmus. The fundi oculi were negative and there was a slight purulent discharge

from her middle ear, but the wound was firmly healed and the mastoid cavity practically healed and the patient's general condition excellent.

I find it very difficult to satisfy myself just what occurred in this particular case, but I think it is probable that the patient had an abscess somewhere in the cranial cavity which was ruptured by the hammering of the skull incident to the mastoid operation. The contents of this flowed back and flooded the areas at the base of the brain producing a marked interference with respiration and heart beat.

I think it probable that this pus did not produce a true meningitis, but that it was probably one of those old sterile abscesses which we occasionally find and the sterility accounts for the fact that we got no growth from the smear ,although diplococci were found present in the fluid.

I admit that this is a very far fetched hypothesis, but I see no other way of accounting for her symptoms immediately after the mastoid operation.

I have trephined and opened the dura in a great many cases of acute lepto-meningitis, but have never yet failed to see one where the increase of cerebral pressure did not immediately start a hernia of the exposed brain as soon as the dura was opened. I base my rejection on the diagnosis of meningitis entirely upon the absence of intracranial pressure during the operation.

Whatever the diagnosis may be, I think there is very little doubt that the patient at the time of operation was moribund and none of the Staff at the E. E. I. who saw her, considered that she could possibly live over three or four hours and I myself considered it more than probable that she would die during the etherization, so I think we may safely ascribe her recovery, whatever the diagnosis may be, to the operation performed.

Discussion.

Dr. Edward B. Dench, New York: In the early '90's I operated on a case for brain abscess. I did not find the

abscess but put in a drain anyway, the man got well and was well ten years later. In regard to meningitis operations, I spoke at the International Congress at Buda-Pesth in 1909 and this procedure was mentioned. A large proportion of the cases get well when the primary focus is removed. A single exposure of the dura is often sufficient. We get no decompression by exposure of the dura, but the cases get well. I have had cases with turbid fluid and a cell count of 1200, yet these cases recovered after simple exposure of the dura. I did an operation on a case with meningeal symptoms and blindness. The boy came in with a recurrent mastoid, and he got well after exposure of the dura. I wish to make the point that Dr. Crockett's patients are apparently hopeless yet they may get well from operation. I think it is a mistake for the otologist when there is clear evidence of meningitis, not to take a chance. One can save a certain number by operation, and the rest of the patients will die anyway. By hammering this into the surgeon you can reduce the morality of otitic meningitis.

Dr. Hugh B. Blackwell, New York: Dr. Crockett did not say anything about stiff neck and disturbance of speech. The involvement was on the left side. This illustrates a point which has been mentioned. When you discover fistula in the internal ear it would seem rational procedure to do a labyrinth operation of some sort. It would seem justifiable under those conditions. The traumatization of the cavity in neglected cases, such as by packing, burning, cauterization, will frequently cause a cavity, and this illustrates the importance of opening the labyrinth at the time of operation. The patient would then escape secondary symptoms.

Dr. G. E. Shambaugh, Chicago: As regards the question of fistula, I am not clear that I got Dr. Crockett's meaning. Did the examination disclose the existence of a labyrinth fistula or was the labyrinth dead? If the test disclosed

a labyrinth which was still functionating, one of course would avoid at the operation anything which might cause the circumscribed labyrinthitis to become diffuse. It is not possible always to find the fistula during the operation.

Dr. B. Alex. Randall, Philadelphia: When a noted foreigner especially interested in labyrinth evisceration visited my clinic, I had two mastoid cases and I had him operate on one. I carefully reserved for myself the one that had labyrinth symptoms—I thought that she was safer in my hands. She recovered completely without labyrinth operation. In cases with marked labyrinth symptoms I clear away any tissue about the fistula which is necrotic and make no attempt to disturb the membranous labyrinth. One had better be conservative in these cases, although labyrinth suppuration often causes cerebellar abscess.

Dr. Wells P. Eagleton, Newark: I am obliged to answer all these questions. These gentlemen have had suppurative meningitis cases with organisms free in the spinal fluid. I instructed Dr. F. Robbins in New York to obtain for me all the cases of suppurative meningitis with organisms free in the spinal fluid, that recovered. We found all these cases were not suppurative meningitis at all. They were protective meningitis. Dr. Crockett did not state whether the organisms were free or inculture. If not free they did not belong to the type one is looking for. I went to the Boston Library to look up all the available information and found only one clearly written reference on the subject. In regard to Dr. Crockett's present case,—with 20,000 cells in the spinal fluid, and a culture of diplococcus in the smear, with the fluid negative, there is no such case. Didn't this woman have epidemic spinal meningitis? That is why it had no cells. The symptoms did not come from the ear at all. I have never heard of 20,000 cells where the case recovered. If he opened the labyrinth and then did a local operation he had exactly the condition favorable. He had

a suppurative process that had already lighted up a protective meningitis at the time of infection of the cerebrospinal system by the micro-organisms. These cases get well. I would like to ask one question. I do not see why the local operation was done. The case was brought to the hospital quickly, the examination was made and the local operation performed. Did it show meningeal symptoms at that time?

Dr. Crockett (closing: I will answer Dr. Eagleton first: I think it was a matter of hospital technic. The case was not properly looked over, although the house surgeon was a very good man. The case should not have had a local, it should have had a cerebral operation. I think those things happen in all big hospitals. In regard to Dr. Eagleton's diagnosis,—I had a hesitancy in making a diagnosis on this cases. I took Dr. Dwyer on one side and got his opinion, and told him how we made the culture. We took a single smear from the fluid and cultured the test tube. He said I had a live diplococcus, but that one must use one or two ccs. of spinal fluid. As I understood him, he said these organisms are partially neutralized by the bactericidal action of the spinal fluid and they are not very active. It takes plenty of fluid. That was probably the trouble with the technic. In regard to opening the labyrinth,—I think that they were entirely wrong in this particular case. I think there was too much brilliant operating. They pretty nearly killed the patient. If they had done the labyrinth operation as well the patient could not have stood it. I think there was too much done as it was. In regard to Dr. Shambaugh's remark, I pretty nearly always agree with him, but in this instance I take exception to his view. In answer to Dr. Dench, with regard to cerebral surgery I am radical, but in labyrinth surgery I am conservative. Whether the patient is alive or dead I would not do labyrinth operation. I believe the mortality would be less if you left the labyrinth alone. The recovery rate will be due to one or two things—either to the internal

protective mechanism of nature, walling off the meningitis. If the meningitis is already set up, the labyrinth operation is shutting the stable door after the horse is stolen. It does not accomplish anything for the good of the patient. That is a very conservative point of view in regard to labyrinth surgery. When that operation first was done, I analyzed the cases. I thought I hastened the death of the patient. I think the Boston men will bear me out in this opinion. It does not make any difference to me whether the labyrinth is alive or dead. In this case the patient was nearly dead herself, but it would not have influenced the operative procedure at all. It was a question of the most logical procedure to be followed. In another successful case the patient was nearly dead and at dehiscence of the otitis of the tympanum. I did trephining and opening the dura with decompression. Four weeks later a local mastoid was done. I considered it a wise thing to do a decompression first and then later the local mastoid. I have followed that procedure with success. I think it is logical. I think the reason the otologist does not have success is in the cerebral surgery. He treats the case primarily as an otologist and secondly as a surgeon. He does the radical mastoid first, and later does the cerebral operation through the mastoid cavity. This is a mistake. That is an error of the otological surgeon. He must get away from that. The cerebral operation should be done entirely independent of the mastoid condition.

LIQUID MOTION IN THE CANALS OF THE EAR.

By PAUL R. HEYL, Ph. D., Bureau of Standards, Washington, D. C.

In November 1921 the Bureau of Standards was asked to give its opinion regarding the possibility of liquid motion taking place in very small tubes, such as the canals of the ear, under conditions of acceleration such as might be produced by revolving the subject in a chair at the rate of one revolution per second or less. The question being one in controversy among physicians and physiologists, and being in essence physical, the Bureau regarded the matter as a proper subject for its consideration.

It has long been known that the labyrinth of the inner ear is concerned in some way with the preservation of bodily equilibrium, and that sea-sickness and vertigo have their origin in disturbances to which the nerve system of the labyrinth is subjected. The great development of aviation during the war brought this subject into prominence. Thousands of candidates for a pilot's license were subjected to the Barany revolving test to determine the adequacy of their sense of balance, or what is commonly called among aviators the ability to "feel the ship".

As an outgrowth of the vast amount of practical work that was thus done it developed that there was no general agreement among physicians and physiologists as to the exact mechanism of this equilibrating sense. Though the point may be regarded by some as one of purely theoretical interest, it was indisputably a point in which our knowledge lacked completeness. And, as it will be seen later, it turned out that the answer to this question unexpectedly proves to be of practical value in certain surgical operations.

The prevailing view among physicians up to the close of the recent war was that the endolymph filling the membranous labyrinth was caused to move or circulate in the canals by the bodily motion of the subject, and that

this circulatory motion stimulated the nerve endings in the labyrinth, giving rise to the characteristic nystagmus of the eyes and to the past-pointing and falling reactions. That a stimulus of the labyrinthine nerve endings was competent to cause these bodily phenomena was well understood. The difference of opinion was as to the mechanism of this stimulus.

Probably nothing contributed more toward the general acceptance of this circulation hypothesis than the relative position of the canals themselves. Three in number, and arranged nearly in mutually perpendicular planes, their orientation suggested that they in some way resolved an impulse into its components. So obvious was this suggestion that those who upheld the circulatory hypothesis were prepared to go to any length necessary in the way of assuming extreme sensitivity for the nerves involved in the perception of what they admitted must be, from the size of canals and the moderate accelerations involved, a circulation of very small magnitude.

The revolving chair test, as usually applied, consists of not more than 10 revolutions in as many seconds, followed by a sudden stop. There is produced in consequence, in normal subjects, a nystagmus of the eyes and a sensation of dizziness lasting for about 20 seconds. The diameter of the canals of the membranous labyrinth is a minor fraction of a millimeter, and the diameter of the semicircle of the canal is usually less than one centimeter. The opponents of the circulation hypothesis, beside denying the possibility of the small acceleration ordinarily used producing a lasting current of 20 seconds in so small a tube, added to the force of their argument by pointing out that the canals of either ear were eccentrically located with reference to the usual axis of rotation (the spinal column) the average distance from the center of rotation being something like four or five times the diameter of the semicircle constituting the canal, and that consequently any tendency to produce circulation in the canals must be of a small differential

order. Constructively, they pointed out that this eccentricity in rotation was excellently fitted to produce inertia effects, resulting in changes of pressure or tension in the membranous labyrinth.

In addition to the revolving chair test, physicians were accustomed to applying a douche of warm or cold water to the outer ear, which produced a nystagmus similar to that produced by rotation. This was explained by the supposition that circulatory convection currents were set up in the labyrinth of the inner ear. There being always a lapse of a considerable fraction of a minute between the first application of the douche and the appearance of the eye-reaction, a time sufficient for the conduction of heat from the outer to the inner ear, but much greater than that of the velocity of any known afferent and efferent nerve action, the circulation hypothesis received additional support from this phenonenon, as no acceleration was involved in the test, and therefore no question of inertia and pressure could arise.

Let it now be stated that the Bureau of Standards has been able to contribute nothing toward a solution of the controversy. The solution, as will shortly be seen, has been reached by the work of the physiologist Maxwell[1]. The present report is largely a resumé and digest of work which has been done by others on the subject; but there is added to this an account of some experiments the results of which confirm the views of those who maintain that circulation in any perceptible degree is impossible. Considerable space has been given to the discussion of the work that has been done by Maxwell and others, as such a resumé is not without value to the busy practitioner.

Experimental work has not been lacking on this question; but until the past three years the results of such experiments have been contradictory and confusing. Two main lines of experiment have been followed: to construct models of the labyrinth and attempt to observe motion of viscous liquids with which they were filled; and direct experiment on animals by elimination of either the labyrinth itself

or the severing of the nerve concerned in the receipt and transmission of the stimulus.

Models of the labyrinth have been constructed and studied by G. Rossi[2]; Marcus Maier and Hans Lion[3]; S. S. Maxwell and W. F. Hoyt[1b]; and by the Bureau of Standards. In the case (2) a positive result was reported; in case (3) it was merely claimed that the presence of such motion (arising from acceleration) was "a safe assumption"; in the case of Hoyt's experiments and in those of this Bureau a negative result (for the customary accelerations) was reached.

Equally contradictory were the results reported in direct experiments on animals previous to the work of Maxwell. Reference to these experiments are to be found in Maxwell's articles (1).

In Maxwell's articles, to which reference has been made, there are described experiments on fishes from which the labyrinthine system had been extirpated in whole or part. The following results were obtained:

1. Both the static and dynamic functions of equilibrium continue after complete removal of all the semicircular canals and all the ampullae.

2. Both static and dynamic functions of equilibrium continue after removal of the otoliths only.

3. In each of the foregoing cases the reactions were noticeably weaker than when both systems (otoliths and ampullae) are unimpaired in function. The two systems therefore reinforce one another independently.

4. Destruction of both the semicircular canal system and the otolith organs completely abolishes all compensatory movements and equilibrium reactions of labyrinthine origin.

5. The various reflex eye movements accompanying bodily rotation can be produced by applying pressure or tension to the nerve regions of the membranous labyrinth.

In brief, Maxwell has definitely located the mechanism of equilibrium in the enlarged portions of the labyrinth rather than in the constricted canals, and has shown that the

apparatus concerned with equilibrium is provided in duplicate independent forms. The nerve stimulus in question may arise either from circulation in these enlarged portions or from change of tension produced by inertia-displacement under acceleration.

It has been pointed out by Dr. Edward G. Seibert of Washington, D. C., that such a duplication of equilibratory apparatus as announced by Maxwell is a fact of importance in certain operations in the region of the ear. The surgeon may hesitate to push the operation as far as conditions indicate as desirable if he fears injuring the canals and thereby destroying the sense of equilibrium; but, if Maxwell's conclusions are confirmed, this fear is groundless.

Other experimenters have extirpated the canal system with contradictory results. Maxwell points out a possible source of this confusion. He began recording the results of his experiments immediately after the removal of the canals, and continued his observations, in some cases, for two days. He found that the sense of equilibrium, nearly normal immediately after the operation, gradually diminished, and in one case had practically disappeared in 24 hours. By the second day this fish, being in a moribund state, was killed for autopsy, which disclosed blood clots in each vestibule. Had he assumed that on account of shock observations made on the day of operation would be unreliable, and had he waited until the following day, it would have appeared that the loss of the canals abolished the compensatory movements, which is obviously not true; for when immediately following the destruction of an organ a function is clearly retained it is indisputable proof that at least that organ is not the only one which can perform the function.

For the precautions taken against retinal stimulation the original papers should be referred to.

With reference to the question which naturally arises as to the specific function of the canals, especially in view of their suggestive spatial arrangement, Maxwell remarks that it is possible that the canals may serve as equalizers

of liquid pressure in a way analogous to the function of the Eustachian tube in equalizing air pressures; that, indeed, if the ampullae were merely diverticulae from the vestibule it is conceivable that pressure conditions could arise in them which might seriously affect their functioning.

Maxwell refers also to the nystagmus produced by heat and cold stimulation, and remarks as follows:

"The fact.....can be explained perfectly without assuming an unbelievable flow of liquid in the semicircular canals. If the temperature difference can cause convection currents in the inner ear at all under the conditions existing in such an experiment, it is certainly more reasonable to suppose that such movements of convection would arise in the mass of liquid contained in the vestibule than in the much smaller space of the canals where the friction would more readily overcome the tendency to movement. These considerations would not in the least invalidate the diagnostic use which Barany has made of the phenomena, but they do supply a rational explanation of its causation."

This excellent work of Maxwell leaves little to be done except in the way of confirmation. This the Bureau of Standards is not equipped to do, either as regards personal or material facilities, as far as physiological experiment is concerned. It has been possible, however, to confirm the non-existence of circulation in the canals, and a brief description of these experiments may not be uninteresting.

Into the face of a piece of glass of the length and breadth of a microscope slide, but about half a centimeter thick, there was ground (and polished) a small circular channel 7 mm in depth. At one point of this channel there was a ground and polished enlargement or vestibule 5 mm diameter and 3 mm deep (Fig. 1). Such a channel corresponds approximately in size to one of the canals of the osseous labyrinth. The canals of the membraneous labyrinth have about one-third the area of those of the surrounding osseous labyrinth, but for convenience of construction and experiment the larger dimensions were adopted. If not circulation is perceptible in canals of this

size, it follows *a fortiori* that none can be assumed in the smaller size.

The enlarged depression was filled with a drop or two of water and a microscope slide was slid in position as a cover, being held in place by the capillary film of liquid between the glass surfaces.

As an indicator of motion it was found that very minute bubbles, about 0.1 mm diameter, served well, if the glass parts were clean. Under these conditions no perceptible adhesion of the bubble for the glass was noticed, the enclosed liquid acting as a sensitive level. By a little practice in sliding on the cover one or two bubbles, as

FIG. 1

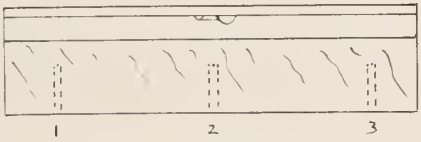

FIG. 2

might be desired, could be introduced, and by careful tilting brought to any desired part of the canal.

The glass plate was mounted on a wooden base, secured to it by wax (Fig. 2). In the bottom of the wooden base were drilled holes 1, 2, 3. (Fig. 2). The hole 2 was centrally located with respect to the canal; holes 1 and 3 at a distance of 3.5 cm. on either side. By means of these holes the apparatus could be mounted on a pivot and rotated by the finger with the canal either centrally or eccentrically situated as desired. With the center of rotation at either 1 or 3 we have approximately the conditions under which the head is rotated in the revolving chair.

With a single bubble in position *a* (Fig. 3) and the pivot in position 2, the system was started slowly in rotation, gradually increasing in speed, and finally given 10 turns in 10 seconds, followed by a sudden stop. No perceptible shift of the bubble was observable in many repetitions of this experiment.

Changing the pivot to position 3, and rotating at one revolution per second, the bubble was seen to move slowly towards the enlargement, that is, toward the center of rotation. This occured no matter in which direction the rotation took place. Repeating the experiment with the center at 1, the bubble moved away from the vestibule

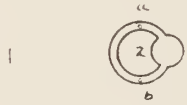

FIG 3

FIG. 4

toward the center of rotation; and again the result was independent of the direction of rotation.

Two bubbles were now introduced and brought by tilting into the positions *a*, *b* (Fig. 3). Rotation about center 3 caused both bubbles to move simultaneously toward the enlargement, finally entering it, and eventually seeking that side of the cavity nearest the center of rotation.

Resetting two more bubbles again in position *a*, *b*, and rotating about center 1, both bubbles moved from the enlargement, met and remained stationary at the opposite side of the canal, from the vestibule, nearest the center of rotation. These results were independent of the direction of rotation, or in other words, signless.

It is evident that we are not dealing here with a circulation, as no circulation can be a signless quantity, nor cause two bubbles to approach each other from opposite sides of a closed circular canal. The cause of these motions of the bubbles is the centrifugal effect of revolution, which tends to throw the heavier substance (water) radially outward, and in consequence force the lighter substance (air bubbles) inward.

Now the various physiological phenomena produced by rotation of the subject change sign with the direction of rotation. It is evident therefore that such phenomena as are observable in the model canal under moderate rotation play no part in causing the Barany reactions; and it may be further stated that no phenomenon capable of causing these reactions is perceptible at the speed of rotation commonly employed in practice.

It is further to be remarked that this centrifugal effect, being radial in direction, in no way interferes with or contributes to the tangential intertia-displacement of the vestibule to which Maxwell ascribes the origin of the Barany reactions.

To repeat these tests under extreme conditions, the water in the apparatus was replaced by mercury, whose density (and inertia) are over 13 times that of water, and whose adhesion for glass is negligibly small compared to that of water. Only enough mercury was used to partly fill the canal (Fig. 4). Rotation about 1 as a center, if the mercury column was symmetrically placed with reference to the line 1, 2, produced no result; but if the column was slightly askew the rotation increased the dissymmetry, finally throwing all the mercury into the vestibule. Rotation about 3, with an unsymmetrical column, restored the column to a symmetrical position as far as possible from the center of rotation.

Rotation about 2 at the rate of one revolution a second for 10 seconds, followed by a sudden stop produced no perceptible shift of the mercury column.

As a result of these experiments with water and mercury, we may conclude that no appreciable circulation of liquid takes place in the semicircular canals of the membranous labyrinth under the conditions of speed and eccentricity as usually applied in medical practice.

BUREAU OF STANDARDS,
(Signed) L. J. B.

REFERENCES.

(1) S. S. Maxwell: Journal of General Physiology: (a) A Comparison of the Effect of Removal of the Otolith Organs and of the Semicircular Canals: 1919, Vol. 2, pp. 123-132.
(b) The Mechanism of the Dynamic Functions of the Labyrinth: 1920, Vol. 2, pp. 349-355.
(c) The Mechanism of the Static Functions of the Labyrinth: 1920, Vol. 3, pp. 157-162.
(2) G. Rossi: Archivio de Fisiologia; Di un modello per studiare gli spostamenti della endolinfa nei canali semicircolari; 1914, Vol. 12, p. 349.
(3) Marcus Maier and Hans Lion: Pfluger's Archiv, 1921, Vol. 187, page 47; Experimenteller Nachweis der Endolymphbewegung im Bogengangsapparat des Ohrlabyrinths bei adaquäter und Kalorische Reizung.

SOME RESULTS OF EXPERIMENTAL WORK ON THE DIFFERENTIATION OF FUNCTIONS WITHIN THE LABYRINTH.

By S. S. MAXWELL, University of California, (By invitation).

In the cartilaginous fishes it is comparatively easy to stimulate or to extirpate different parts of the labyrinth with relatively little injury to other parts; for this reason the selachians are the best animals in which to study the differentiation of functions of the ampullae and the otoliths. These fishes present another advantage in that their compensatory movements in response to rotations in the various planes are extremely regular and precise.

It can be shown in the dogfish that rotation in the plane of any semi-circular canal causes a definite set of movements of eyes and fins, and that the same movements are excited if the ampulla of that canal is artificially stimulated (mechanically or electrically). Thus rotation to the right in the horizontal plane (around the dorsoventral axis of the body) causes both eyes to deviate to the left; stimulation of the right horizontal ampulla causes the same movement. Rotation to the right around the longitudinal body axis, so that the right side inclines downward and the left side upward, causes an upward deviation of the right eye and downward of the left. These deviations are also seen when the ampulla either of the right anterior vertical or right posterior vertical canal is stimulated. But stimulation of the anterior vertical ampulla causes, in addition to the vertical deviation, a backward rotation around the visual axes, while stimulation of the posterior vertical ampulla causes a rotation forward around the visual axes.

The above observations of Lee (1) and others seemed to confirm the hypothesis that the semi-circular canals with their ampullae are the organs of the dynamic functions of the labyrinth and that it is through their stimulation

that compensatory movements are excited. I have been able, however, to remove the ampullae from the dogfish ear without injuring the otoliths, and have found that in the absence of all six ampullae and canals, the normal compensatory movements of the eyes and fins are made to rotations in all planes except the horizontal (2). If now the otoliths also are destroyed the compensatory functions of the labyrinth totally disappear.

Removal of the otoliths without injury to the ampullae is also possible in the dogfish. If the otoliths are removed without injury to the other vestibular structures, normal compensatory motions occur to rotations around the transverse and the longitudinal body axes. The only pronounced difference between the results of removal of all the ampullae and removal of all the otoliths is this, that the animal without otoliths shows normal compensatory motions to rotations in all planes; the animal without ampullae shows normal compensatory motions to rotations in all planes except the horizontal.

In the preceding paragraphs I have said that "normal" compensatory movements occur after loss of otoliths only or of the ampullae only. This statement should be modified by mention of the fact that in both cases the reactions are slower and less vigorous than in the uninjured animal. This raises the question whether in addition to their equilibrium functions the nervous structures of the labyrinth have also tonus effects, and if so do these tonus effects originate in the ampullae as Ewald (3) believed, or in the otolith organs as the work of de Kleijn and Magnus seems to show.

If we remove the three ampullae from one ear of a dogfish, the animal maintains an unsymmetrical position of the eyes and fins, a position which is practically the same as that which occurs after total destruction of one labyrinth. If now the three ampullae are removed from the other ear, the asymmetry disappears. It can be shown from this and other experiments that the ampullae do

exert a continuous tonic effect upon certain muscle groups, and that these effects are unsymmetrically distributed so that, for example, the ampullae of the right ear influence particularly the muscles which elevate the right eye and those which lower the left eye. We can thus definitely affirm the existence of a tonic influence of the ampullae.

If we destroy the otoliths of one ear, only little or no asymmetry of eyes or fins is to be seen. It might be inferred from this that the maculae, unlike the cristae, are without influence on muscle tonus. But the matter is not so simple, as can be seen by comparing stimulation experiments on the ampullae and on the otoliths. When we stimulate the ampulla of the right anterior vertical canal, the right eye goes up, the left eye goes down, and both eyes roll forward on their axes. But we cannot stimulate this ampulla in any way which will cause these movements to be in the reverse direction. Stimulation of a macula, on the other hand, can cause movements in both directions (4). This can be very beautifully seen in the shovel-nosed ray. When we press directly downward upon the right side of the otolith of the decessus utriculi, the right eye goes down and the left eye goes up; when we press upon the left side of the recessus, the left eye goes down and the right eye goes up. The results are the same no matter whether we stimulate the recessus of the right or of the left ear, or if we have first destroyed the otoliths of one ear before stimulating the other. This shows that the connections of the maculae with the muscles are symmetrical, and hence our asymmetrical operations do not prove the absence of tonic influence by the absence of asymmetry of position after destruction of the otoliths of one ear.

Stimulation of the otolith of the recessus utriculi leads to other interesting results. By the manipulation of this one otolith the eyes may be caused to rotate forward on their axes, backward on their axes, right eye up and left down, or the reverse. Since all these movements, move-

ments which are the normal compensations to bodily rotations both to right and left around the longitudinal body axis, and head downward and upward around the transverse axis, can be obtained from this one otolith, there remains no need for the apriori conception that each otolith functions for movements in one direction only or in one plane only. In the dogfish destruction of the otoliths of the sacculus and lagena has no apparent effect on equilibrial functions.

Destruction of the single otoliths does not seem to be possible in the higher vertebrates, but de Kleijn and Magnus (5) have been able by a method first discovered by Wittmaack (6) to cause a general destruction of the otolith organs while apparently leaving the ampullar endings uninjured. Guinea pigs were subjected to extremely rapid centrifugalization causing a disruption of the otolith membrane, which could be demonstrated histologically. These guinea pigs retained the ability to respond to rotations by the usual compensatory motions, but the compensatory positions were not retained. In the latter respect the results differ from my observations on dogfish with otoliths destroyed.

My experiments on the otoliths show also that pressure is not the stimulus for the macula. Not the weight of the otolith, but the tension change caused by the displacement or change of position of the otolith, is the stimulus. Curiously enough, de Kleijn and Magnus (7) have arrived at practically the same conclusion by a totally different method. They have shown that in the rabbit the maximal tonic effect of the otolith occurs not when it presses upon the macula, but when it is suspended from the macula.

This discovery was all the more interesting because I had already found that the mechanism of stimulation of the ampullae depends also upon this principle (8). When the head performs a rotational movement, the inertia of the mass of liquid in the vestibule exerts a pull upon the attachment of the membranous canal to the utriculus, and

the tension is communicated to the crista. This is shown by the fact that after cutting across the utriculus near the entrance of the horizontal canal no reaction occurs to rotation in the horizontal plane, although the ampulla remains as sensitive as ever to mechanical or electrical stimulation. It is a striking fact that cutting the canal between the ampulla and the vestibule puts the ampulla completely out of function, but cutting the canal on the other side of the ampulla does not in the least interfere with its activity. This fact alone would show that the normal stimulus of the crista is not an endolymph current in the canal. Maier and Lion (9) have recently shown that under certain conditions inertia currents can be produced in a model with canals of approximately the caliber of those in the human ear, and they have assumed this to be proof that the adequate stimuli for the cristae are endolymph currents. Their observations do not justify such a conclusion. Furthermore, I have shown that the canals may be cut or plugged or ligatured or they may be artificially moved out of their normal planes without interfering with their functions. The conception of currents as the cause of the excitation of the cristae is not supported by the facts.

Permit me to add in conclusion a general remark. A working hypothesis based upon a priori consideration can be very useful as a program for experimental investigation, but it can be very misleading if drawn upon as "an explanation". The history of research on the labyrinth shows that there has often been an undue dependence on a priori conclusions. In going through the enormous literature of the subject, one is impressed at times with the remarkable unanimity of the results obtained and the conclusions reached in certain geographical regions. We shall proceed more safely and no less rapidly if we seek for facts rather than for explanations. An explanation after all merely states that some facts are similar to or are related to some other facts.

REFERENCES.

1. Lee, Frederic S.: A study of the sense of equilibrium in fishes. J. Physiol, 1894, XV., 311.
2. Maxwell, S. S.: Labyrinth and Equilibrium. I. A comparison of the effect of removal of the otolith organs and of the semicircular canals.
J. Gen. Physiol., 1919, II., 123.
3. Ewald, J. R.: Physiologische Untersuchungen über das Endorgan des Nervus octavus.
Wiesbaden, 1892.
4. Maxwell, S. S.: Labyrinth and equilibrium. III. The mechanism of the static functions of the labyrinth.
J. Gen. Physiol., 1920, III., 157.
5. de Kleijn, A., and Magnus, R.: Ueber die Funktion der Otolithen. II. Mitt. Isolierte Otolithenausschaltung bei Meerschweinchen.
Arch. ges. Physiol., 1921, CLXXXVI., 61.
6. Wittmaack, K.: Ueber Veränderungen im inneren Ohre nach Rotatonen.
Verhandl. Deutsch. Otol. Ges., 1909, XVIII., 150.
7. de Kleijn, A., and Magnum, R.: Ueber die Funktion der Otolithen. I. Mitt. Otolithen bei den tonischen Labyrinthreflexen.
Arch. ges. Physiol., 1921, CLXXXVI., 6.
8. Maxwell, S. S.: Labyrinth and equilibrium. II. The mechanism of the dynamic functions of the labyrinth.
J. Gen. Physiol., 1920, II., 349.
9. Maier, Marcus, and Lion, Hans: Experimenteller Nachweis der Endolymphbewegung in Bogengangsapparat des Ohrlabyrinths bei adequäter und kalorischer Reizung.
Arch. ges. Physiol., 1921, CLXXXVII., 47.

SENSIBILITY OF PATHOLOGICAL EARS TO SMALL DIFFERENCES OF LOUDNESS AND PITCH.

By V. O. KNUDSEN and GEO. E. SHAMBAUGH, M. D., Chicago, Ill.

Introduction.

This is a preliminary report of an investigation undertaken in the Department of Physics at the University of Chicago and which is of interest to otologists. It is an effort to determine the sensibility of the ear to small differences of loudness and pitch. Another investigation undertaken by Mr. Minton in the same laboratory has aimed to determine the minimum audibility of tones. The latter work is to be discussed at this meeting by Dr. Wilson. Both investigations have made use of the method of charting graphically the results which renders the study of their findings more simple.

The primary interest which these researches so far have had for the otologist is the side-light which they throw on the physiology of tone perception. Quite apart from this interest the question naturally arises whether the results of this work may not be found to be of some practical value in solving the clinical problems with which the otologist is confronted. Research is usually undertaken without a thought as to any practical application. Where such application is feasible this fact soon becomes apparent. These researches are not peculiar in that they do not aim to solve our clinical problems. Whether practical application can be made of these tests is a question for the otologist to work out. It is a question of no small importance. Any additional aid in our work is readily welcomed. The question is of special importance since there has been some discussion of a plan for establishing laboratories to which clinicians should send their patients to have these tests made and for which the patient should be charged a fee, somewhat as is being done in X-ray work.

Such a plan if carried out should have the sanction of the otologist.

In order to be able to formulate a conclusion as to a practical application of these tests, it is of primary importance that we have a clear conception of our clinical problems lest we be led into confusing a study which may be purely of scientific interest with something quite different. As regards the clinical problems involved in the hearing tests, the first question is concerned with distinguishing cases where there is some possibility of improvement by treatment from those where from our knowledge of pathology it is quite clear that treatment could be of no assistance. I do not believe that this diagnosis presents any particular difficulty for the experienced otologist. It is ordinarily rather easy to determine this by other simpler methods of examination. I have not been able to make out as yet that its solution is materially assisted by these additional tests. Still it is possible that something may yet be worked out along this line. Another practical use which is made of the functional tests of hearing is for determining what improvement, if any, is resulting from treatment of defective ears. This subject has received a good deal of attention in the past. The conclusion usually reached has been that the voice tests are superior to other methods, mainly because we are primarily interested in securing improvement in the hearing for the spoken voice and because it is well known that there need be no distinct relation between the ability to hear the voice and the hearing for some mechanical device, such as the watch and the various tuning forks. Tests showing the entire tone range should, it would seem, be able to afford some sort of an index of what we aim to get, that is, of the degree of improvement for the spoken voice. It does not seem probable, however, that the testing of the tone range would supplant the testing with the voice for this purpose unless some device should be invented which would render these tests simple enough to be carried out with sufficient readiness to serve this purpose practically.

It has been suggested that these tests are valuable when repeated from time to time for showing changing status of hearing in cases of progressive deafness, such as, otosclerosis or primary nerve deafness. I am inclined to think that with these tests we can get a more satisfactory line on the progress of the hearing condition in these cases than by other methods. It is a question, however, whether we would be justified in advising patients to go through with these elaborate tests from time to time especially since in just these cases there is no practical assistance to be derived from them.

There still exists a great deal of haziness in the minds of many practitioners regarding the clinical problems in otology. To assume that these newer tests of hearing are to bring in a new era in otology in which these tests will be able to dispel the prevailing pessimism that exists regarding the cure of deafness shows a failure to grasp the simple clinical problem presented in these cases. Pessimism does exist exactly as it does in the treatment of locomotor ataxia or in cases where the hair is turning gray for the very good reason that we know from our study in pathology that such conditions as otosclerosis and degeneration of the eighth nerve can neither be improved nor their progress checked by treatment.

These remarks are intended as a warning against a too hasty conclusion on the part of otologists that these tests have a commercial value, in other words, are of practical assistance to the otologist. Since the time of Helmholtz the physicist has played an important part in the study of the problem of hearing. Much of this work has been done in co-operation with otologists. This co-operation, therefore, has long been established. It is to be hoped that something of practical value may come from the fresh impetus arising from these newer tests. What we may expect quite probably will not be related to our primary problem, that of diagnosing the treatable from the incurable form of deafness or much less to dispelling existing pessimism regarding the incurable forms of deafness but

may rather be along the lines of improvements in the devices intended for bridging the chasm between defects in hearing and the normal ear. It would seem that a study of the minimum audibility of tones in the several parts of the tone range and of the sensibility of ears to small differences of loudness and pitch may furnish data which could be turned to a practical purpose in the construction of improved hearing devices, especially the electrical hearing devices, which, imperfect as they now are, have already gained a wide application. Until it becomes apparent that these tests add something essential to our methods of diagnosis, something which simpler methods do not give, it would be a mistake to lend encouragement to the idea that this work has a practical value for which the patient can be asked to pay.

The report of Mr. Knudsen follows:

The writer has recently presented a paper before the American Physical Society on "The Sensibility of Normal Ears to Small Differences of Loudness and Pitch[1]." The work reported in the present paper is an extension of that investigation applied to pathological ears. A knowledge of the sensibility of the ear to small differences of loudness and pitch is essential to intelligently prescribed artificial aids for hearing. If a pathological ear is normal in its pitch and intensity discriminating power, appropriate amplification of the sounds coming to the ear will restore normal hearing. But, if the affected ear is seriously subnormal for these functions, amplification is not adequate for the restoration of normal hearing, since the interpretation of speech and musical sounds by the ear requires the capacity to appreciate small differences of pitch and loudness.

The method of just discernible differences is employed in the present investigation for determining the sensibility of the ear to small differences of loudness and pitch. The source of sound used is a telephone receiver actuated by

1 Sensibility of the Ear to Small Differences of Intensity and Frequency—V. O. Knudsen, Abstract in Physical Review 19, 261 (1922).

energy from a vacuum tube oscillator. The oscillator will produce tones of any desired frequency between 30 d. v. and 20,000 d. v. By means of a divided resistance circuit

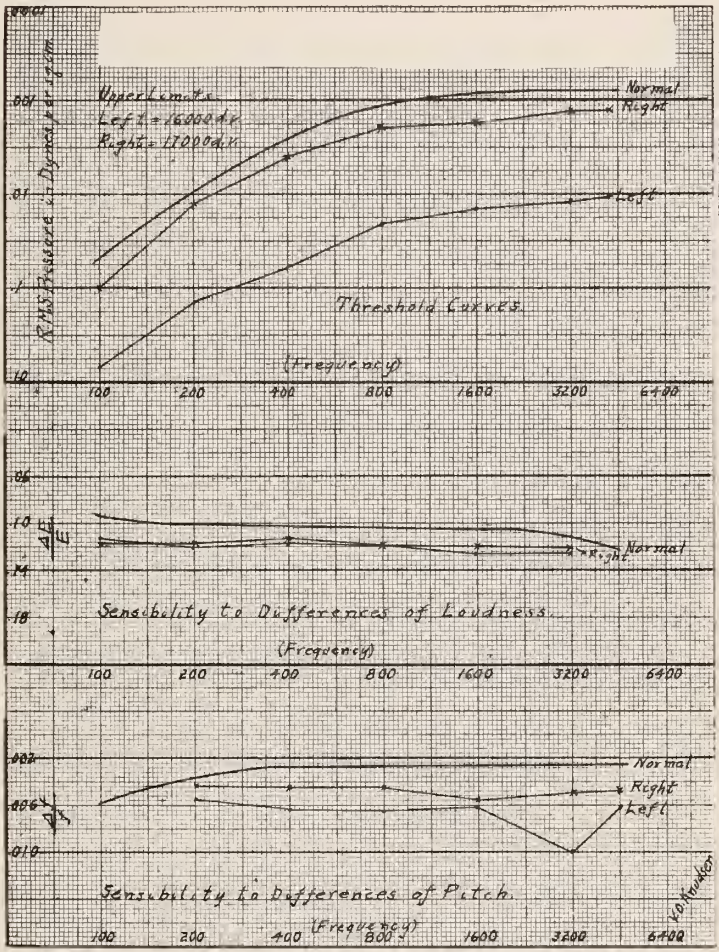

Fig. A. Case of Vertigo; Probably of Intracranial Origin, Hearing for the whispered voice normal. Diplacusis.

the intensities of the tones can be varied by any desirable and measurable intervals from the threshold intensities up to intensities sufficiently loud to excite the sensation of pain.

The circuit is designed in such a way that a motor controlled key periodically changes, by any desired intervals, the resistance R across which the receiver is connected.

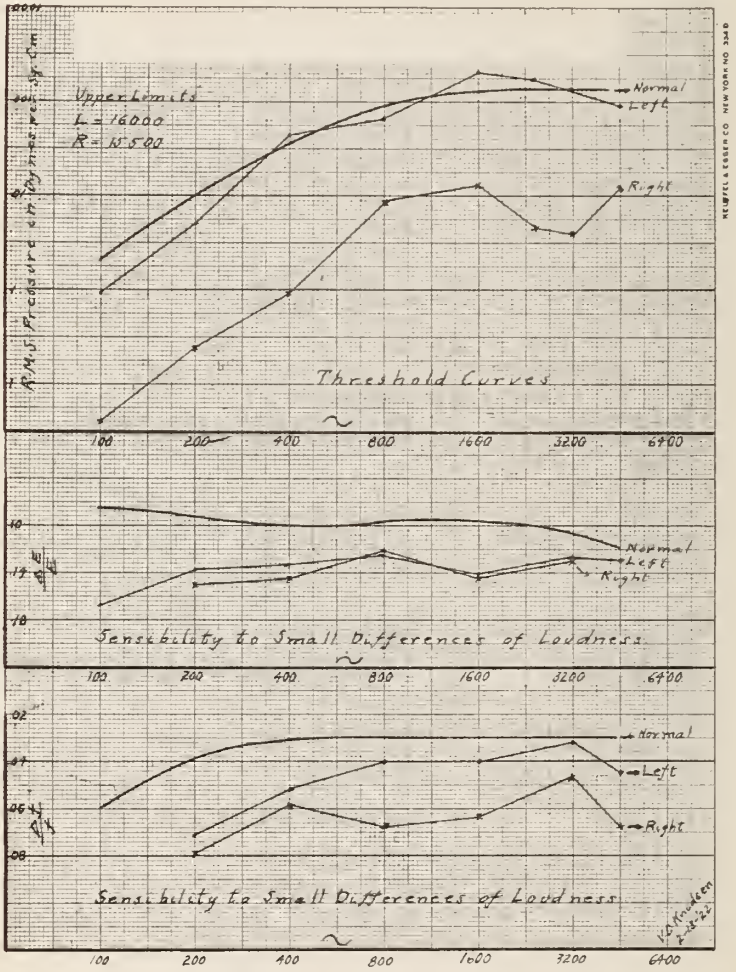

Fig. B. Case of Degenerative Changes in the Labyrinth Diplacusis.

The tone emitted by the receiver will therefore periodically and abruptly fluctuate from a tone of one loudness to a tone of greater or lesser loudness. The two tones are

of equal duration and alternate at a rate of about 50 per minute.

If the difference of loudness of the two tones is greater

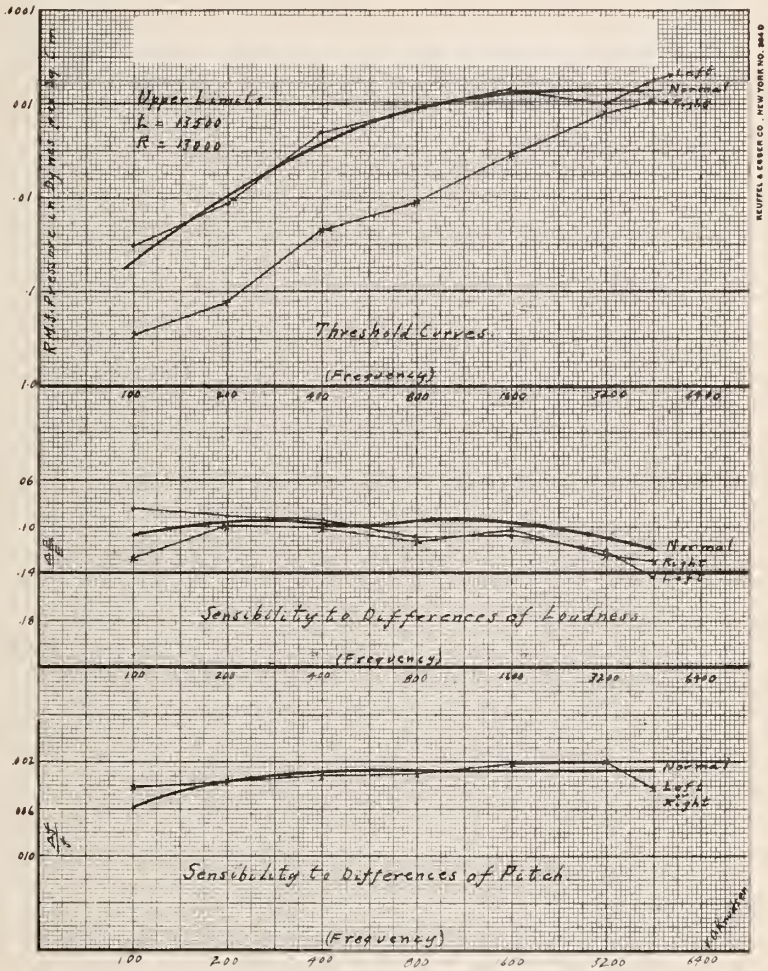

Fig. C. Case of Otosclerosis Incipient.

than the smallest perceptible difference for the ear under test the two tones will be heard as a flutter tone, otherwise they will be heard as a steady tone. Hence, to determine

for a particular ear the smallest perceptible difference of loudness it is only necessary to properly vary a single wire resistance until the person under test decides when the

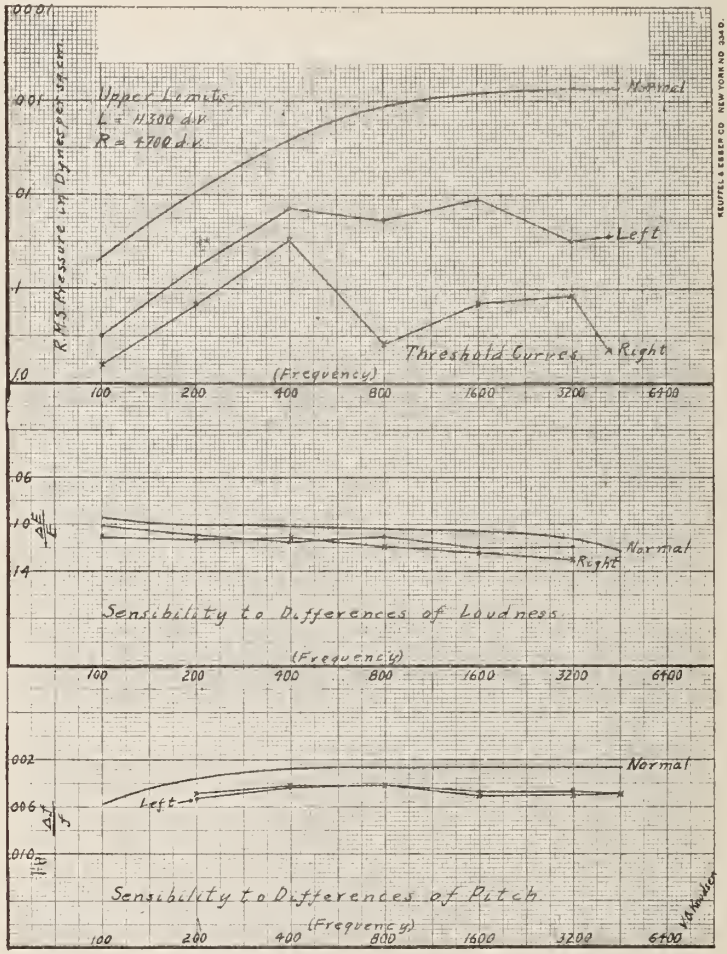

Fig. D. Case of Nerve Deafness. Degeneration in the Labyrinth.

flutter appears as the difference of loudness of the two tones is increased from zero, and when the flutter disappears as the difference of loudness of the two tones is decreased from a plainly perceptible difference.

A similar procedure is used for determining the smallest perceptible difference of pitch, except that the frequency instead of the intensity of the tone emited by the receiver

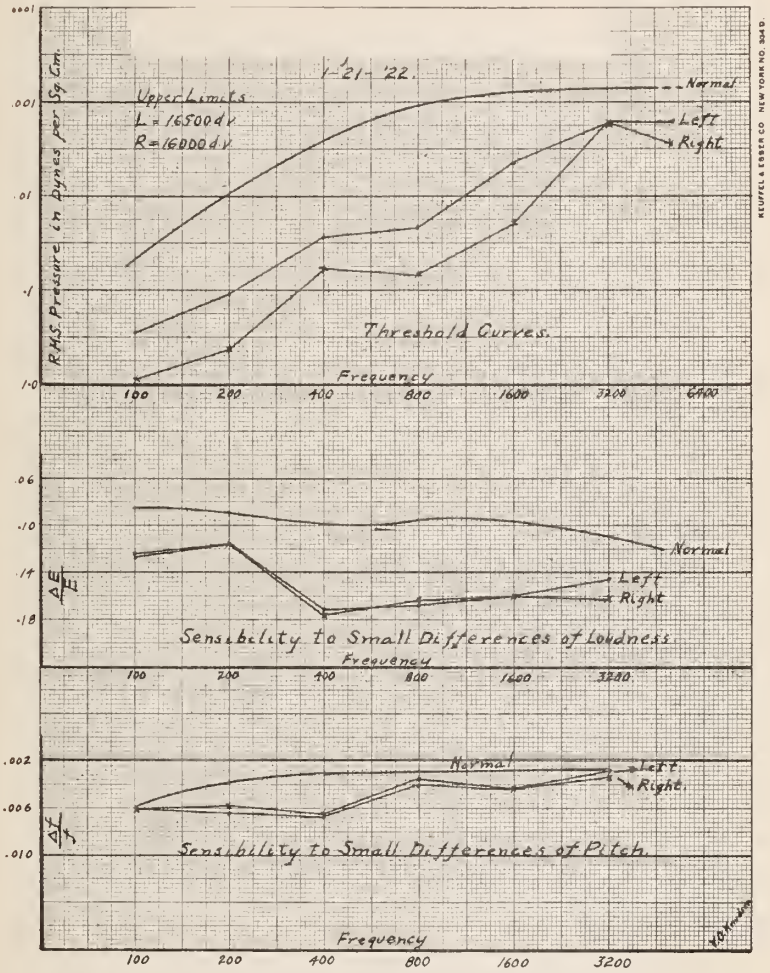

Fig. E. Case of Otosclerosis.

is made to alternately fluctuate from a tone of one pitch to a tone of higher or lower pitch.

The next three slides will show some recent sensibility curves of normal ears. The first slide shows how the

sensibility of the ear to small differences of loudness depends upon the intensity of musical tones. $\Delta E/E$ is the ratio of the smallest perceptible difference of energy of

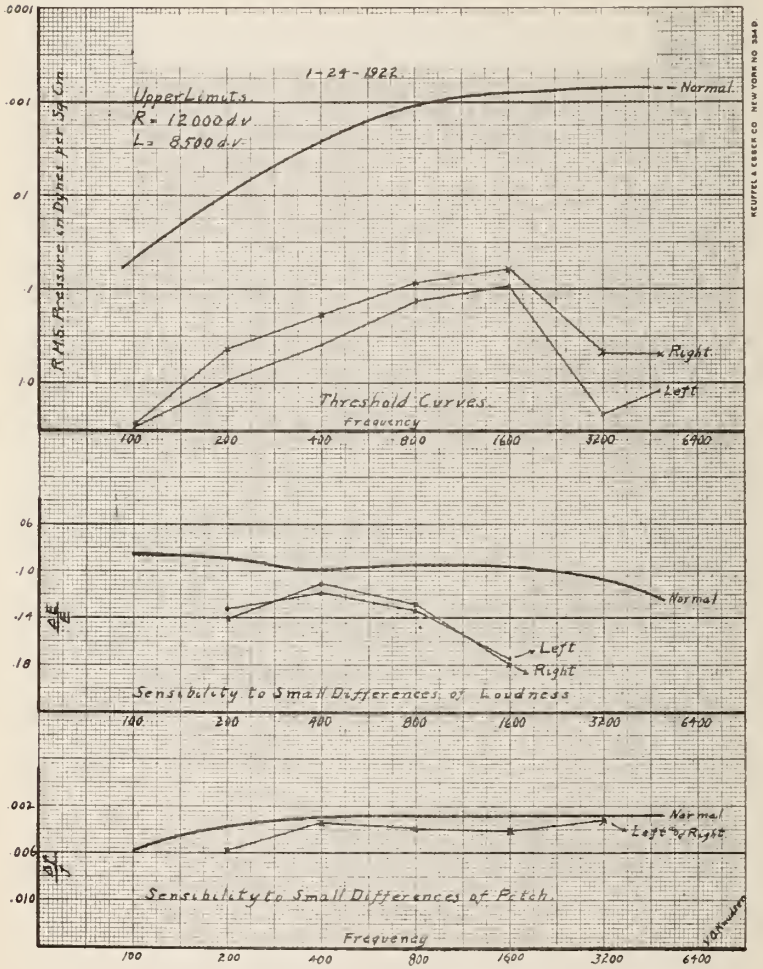

Fig. F. Case of Otosclerosis. Advanced.

a tone to the total energy of that tone. The curves show that the normal ear can perceive smaller percentage changes of loudness for moderate and loud tones than it

can for feeble tones. For moderate and loud tones the normal ear can perceive a difference in the energy of a tone of about 10%. This means that a normal ear can distinguish

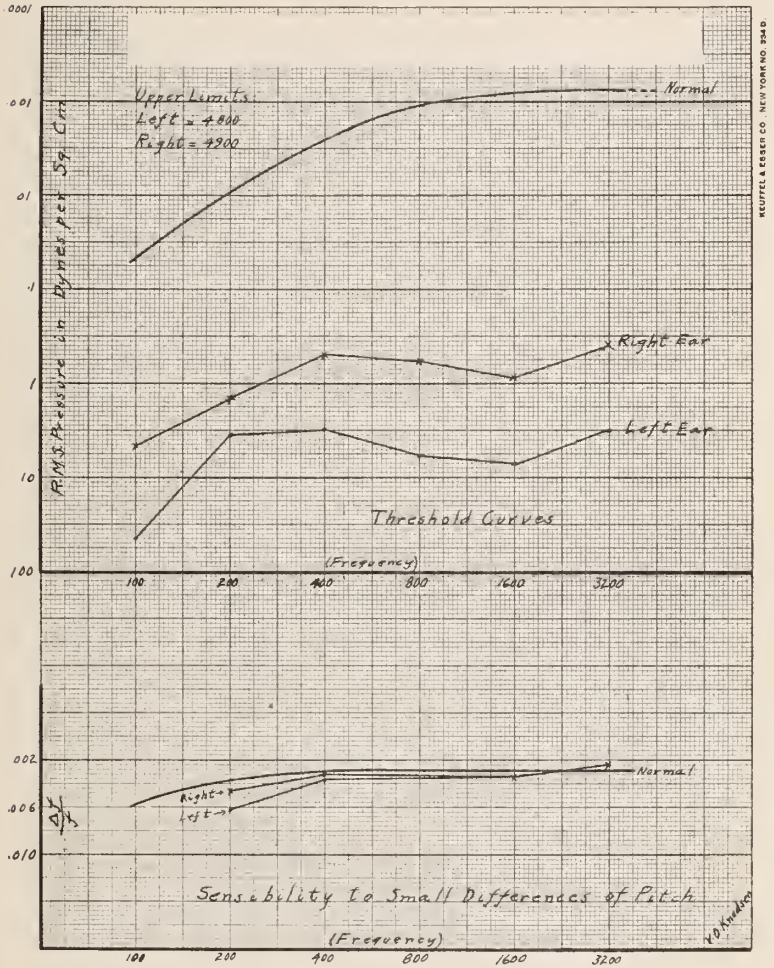

Fig. G. Case of Otosclerosis. Advanced.

about 400 graduations of loudness for tones of medium pitch.

The second slide shows how the sensibility to small differences of loudness depends upon the pitch of musical

tones. The individual curves of 19 ears and the average curve of the 19 ears are shown. There are noticeable individual differences, but the average curve indicates that

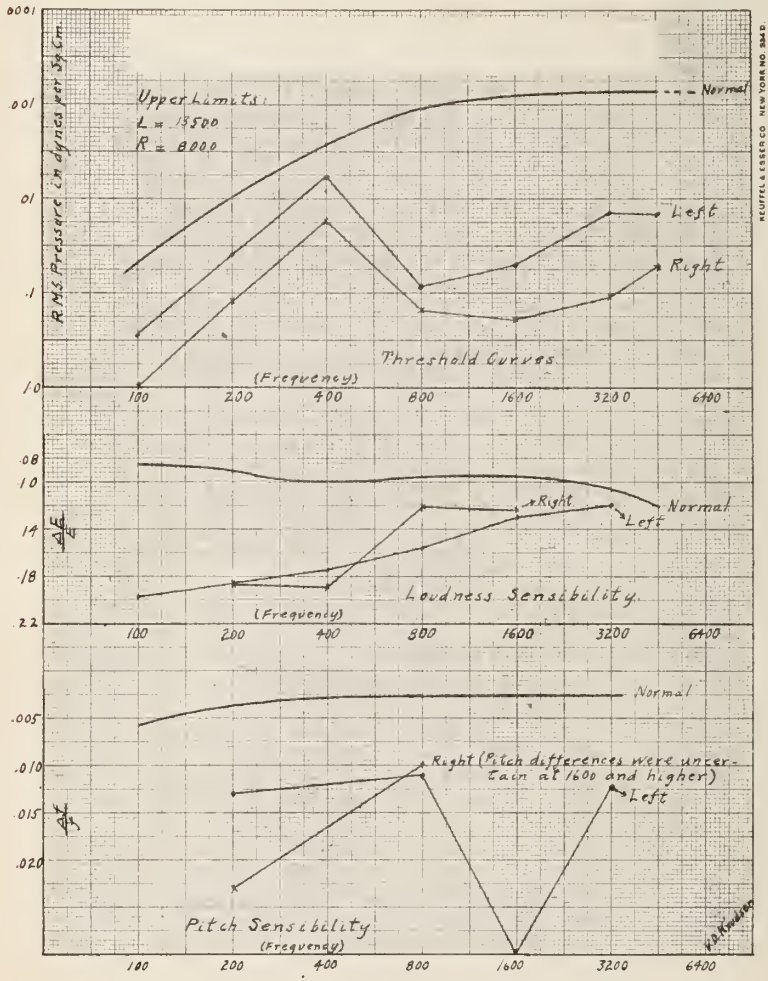

Fig. H. Case of Nerve Deafness.

the sensibility to differences of loudness is almost independent of the pitch over the range used in speech and music.

The third slide shows how the resolving power, i. e. the sensibility to small differences of pitch, varies with the

pitch of musical tones. $\Delta N/N$ is the ratio of the smallest perceptible difference of the frequency of a tone to the whole frequency of the tone. These curves are similar to those obtained by C. E. Seashore and his assistants at the University of Iowa. They show that the ear can perceive smaller musical intervals for moderate and high tones than it can for the graver tones. The average curve shows that at the higher tones the normal ear can perceive a difference of pitch corresponding to about 1/20 of a semitone. The normal ear can therefore distinguish about 2,000 gradations of pitch within the audible range (20 d. v. to 20,000 d. v.)

The sensibility characteristics of a number of pathological ears recently examined are indicated by the following figures. Each figure consists of curves which quantitatively compare the response characteristics of the ear under test with those of the normal ear. The upper curves compare the threshold intensities of the affected and normal ears. They show simply how much louder the tone must be for the affected ear than for the normal ear. The ordinates give the root mean square pressure in dynes per square centimeter in the external ear cavity. This pressure is proportional to the amplitude of vibration of the tonal stimulus. Thus to produce a barely audible tone for A's left ear at 100 d. v. it was necessary to have the amplitude of vibration about 16 times as great as the amount required for a normal ear. The middle curves compare the sensibility to small differences of loudness of the affected and normal ears. $\Delta E/E$ is the ratio of the smallest perceptible increment of energy of a tone to the total energy of the tone. The lower curves compare the sensibility to small differences of pitch of the affected and normal ears. $\Delta F/F$ is the ratio of the smallest perceptible difference of frequency of a tone to the frequency of the tone.

The curves for the cases presented in the above figures represent the response characteristics of ears affected by varied types of fixation deafness, or nerve degeneration, or diplacusis, or combinations of these. All of the curves

indicate that these pathological processes do not greatly affect the pitch and intensity differentiating mechanisms of the ear. In spite of the advanced nerve degeneration in cases "F" and "G" the pitch and intensity differentiating capacities were almost normal. Only one case has been observed in which the pitch discriminating power was noticeably sub-normal, and in that case it was not serious.

Interesting data on diplacusis are accumulating. Thus far seven cases have been carefully examined. In some cases the affected ear hears too low, in others too high. In some cases the affect extends over the entire audible range of pitch; in others, it is restricted to only a portion of the tonal range. The displacement of pitch identification in the seven cases examined varies from ¼ tone interval in some instances to slightly less than an octave in one other instance. In the case of B, the right ear could not associate the property of pitch with tones whose frequencies were below 600 d. v., yet his response curves indicate that the affected ear could perceive differences of pitch and loudness with nearly the same facility as a normal ear. He said, "A change of pitch seemed like a change in the quality of a sort of a wind sensation in the right ear."

In a subsequent paper the writers hope to give a more complete report of their study of diplacusis, together with the bearing of their data on theories of audition.

THE STUDY OF THE TONAL RANGES IN LESIONS OF THE MIDDLE EAR.

By L. W. DEAN, M. D. and C. C. BUNCH, Ph. D., Iowa City, Ia.

The research work which we are reporting represents a large amount of careful, conscientious work especially on the part of Mr. Bunch, who either made or supervised all the functional tests of hearing which are indicated. This report is confined to the examination of the auditory perception of a large series of cases of so-called middle ear trouble. In all cases exhaustive clinical examination was made. These examinations were grouped and studied and the results are here summarized and presented for your consideration.

PLATE 1 is the diagram of the clinical record made for each case examined. In only a few cases where it seemed desirable to have specific information regarding the vestibular functions were the turning tests made.

The hearing for whispered and spoken voice was first determined. Next the pitch range audiometer was used to determine the audition for tones between 30 and 7070 d. v.

The records with the audiometer are easily indicated by means of a graph shown in the plate. The pitch of the tones is indicated at the bottom and the loudness of the tone by the heighth of the curve on the graph, no tones at all being normally heard at zero. The curve shown on the graph is the field of hearing for a normal ear.

In the curves of cases studied that we are going to show, the curve was secured by testing the audition of the ear examined for every tone from 30 d. v. to 7070 d. v. The curve thus secured compared with the normal curve which is stamped on every chart gives an approximate idea of the relative hearing power for these tones.

Notes:

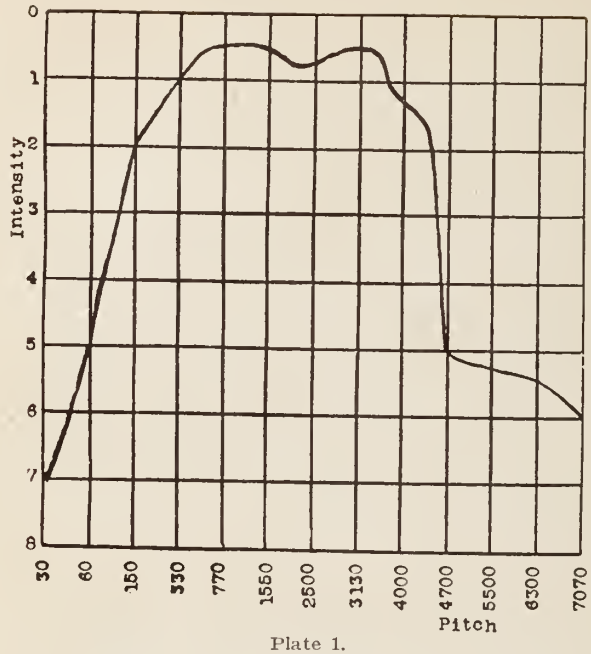

Plate 1.

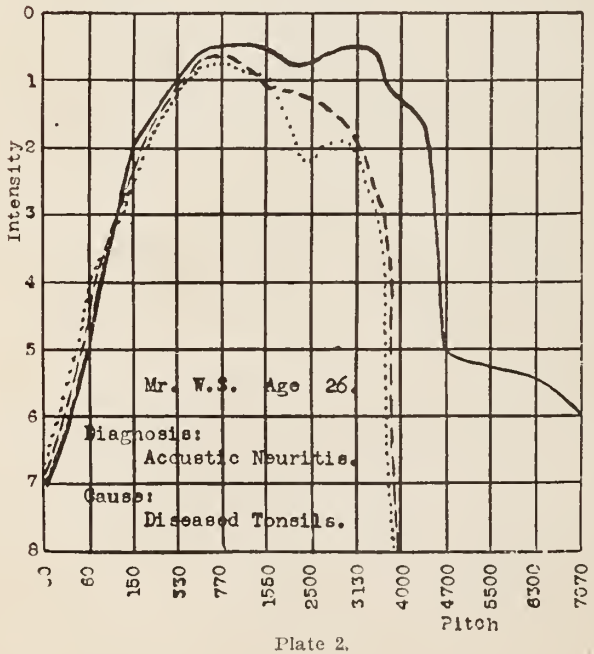

Plate 2.

If, for instance, the curve at 1,000 d. v. is only half as high as normal, this does not mean that hearing for that note is one-half normal but only that hearing for the note is markedly decreased. We are not justified in assuming that the curves represent the exact audition for the note that is being tested. The fractions which express quantitatively the audition for various notes, when this is determined with the Bezold forks, are likewise very inexact. As the intensity of sound varies with the square of the distance, with the square of the amplitude of the vibration, and is influenced by the rapidity of vibration of sound it is an extremely difficult matter to determine in any way the exact amount of audition for any given note.

If the curve of the ear being examined should show a tone gap in the tonal range, this does not necessarily mean that there is an absolute tone gap but it does indicate that the ear is unable to perceive the loudest tones capable of being produced by the audiometer. These gaps found by the audiometer have been verified in the psychological laboratories by tests with organ pipes and other instruments, producing pure loud tones. It is easy to conceive that if a note could be produced of greater intensity than that which the audiometer can give it might be heard when the audiometer would produce no sensation of sound.

In the following charts the normal curve is indicated by a continuous line; curves for the right ear by dashes; and for the left ear by dots.

For the benefit of those who are not interested in the audiometer readings we have made in each case a quantitative determination of the audition for the 50 d. v. fork, the c, c^1, c^2, c^3, c^4, and c^5 forks.

The Rinne, the Weber, and the bone conduction tests were made with the 256 fork. In some cases the 128 and 512 d. v. fork was also used. These forks were weighted to minimize the production of confusing overtones.

At the bottom of the plate next to the audiometer curve is a measurement of the upper tonal limit as determined with the Galton whistle, the Koenig cylinders, and the

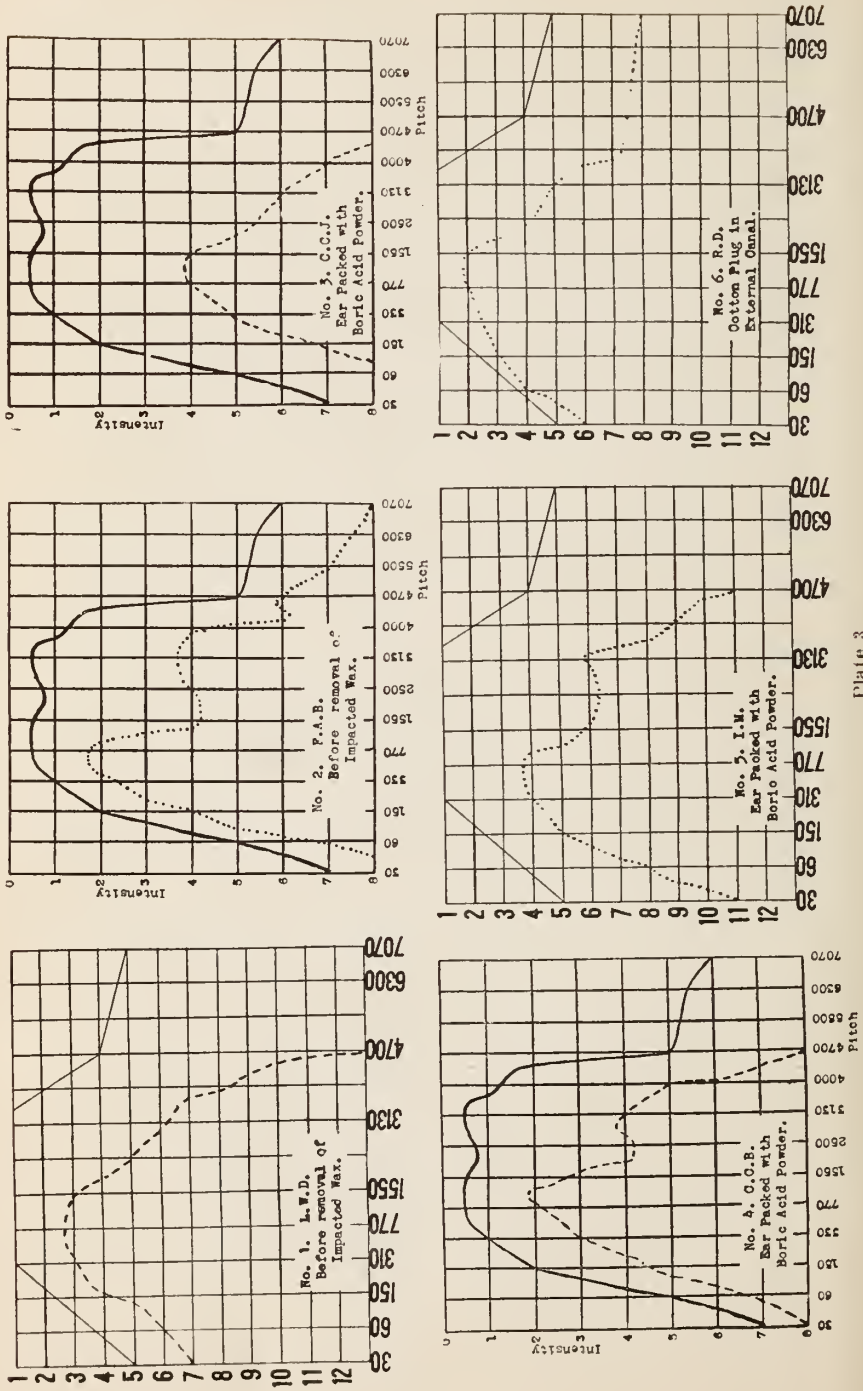
No. 1. L.W.D.
Before removal of
Impacted Wax.
No. 2. P.A.B.
Before removal of
Impacted Wax.
No. 3. C.C.J.
Ear Packed with
Boric Acid Powder.
No. 4. C.C.B.
Ear Packed with
Boric Acid Powder.
No. 5. I.M.
Ear Packed with
Boric Acid Powder.
No. 6. R.D.
Cotton Plug in
External Canal.
Intensity
Pitch

Plate 3

Monochord. The upper limit of the monochord is determined both by A. C. and by B. C. With the Galton whistle which we use the normal upper limit is 21,000 d. v. With the Koenig cylinders the upper limit is 43,960 single vibrations and with the Monochord by A. C. 19,000 d. v., by B. C. 20,000 d. v.

In each case studied we have been interested in determining if in addition to the middle ear lesion there was also present a lesion of nerve or end organ.

Suggestive of a lesion of the acoustic nerve or its end organ is the following:

1. The audition for the higher notes is poorer than that for the lower or middle part of the tonal range. If the lower notes of the tonal range up to about 3,000 d. v. are heard normally and there is a diminution in the audition for the notes in the upper part of the range as is shown by PLATE 2, this diminution of audition for the higher notes is suggestive of a lesion of the nerve or end-organ. If, however, as is shown in PLATE 3, there is a uniform diminution in audition for tones throughout the range it does not indicate a lesion of nerve or end-organ. The curves shown in PLATE 3 are the curves secured from; first, a case of impacted wax in the external canal; second, a similar case; third, three cases of individuals with normal hearing where the external canal has been packed with borac acid powder, and lastly, a case of cotton in the ear which was soaked with cerumen and was in contact with the drumhead. In each of these cases you note the diminution in audition for all tones throughout the range. As we view it, there would have to be a distinctly greater diminution for the notes in the upper part of the range as compared with the tone best heard in the lower or middle part in order to indicate positively a lesion of the nerve or end organ. In some of these cases the c^5 fork was not heard at all.

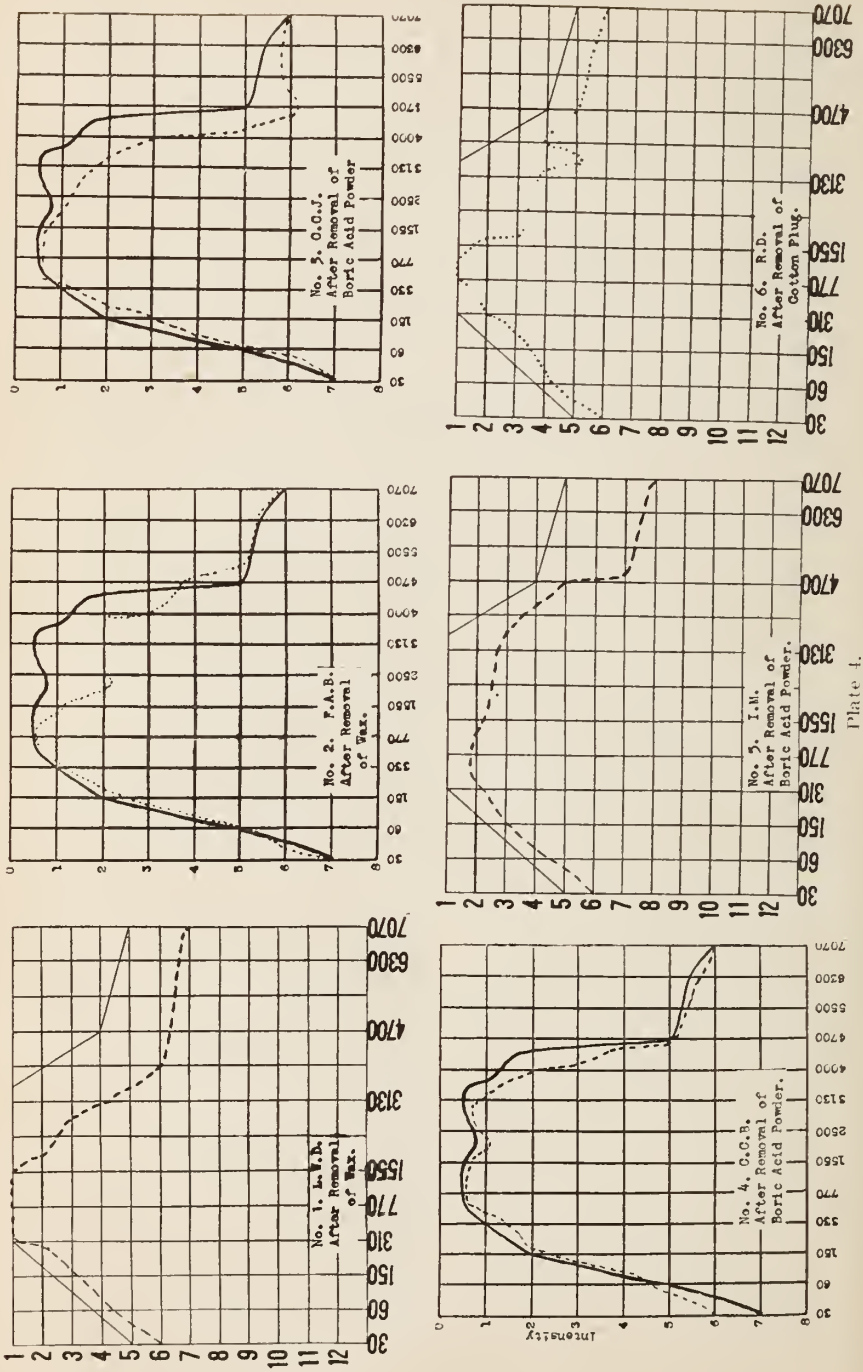
No. 1. L.W.D.
After Removal
of Wax.
No. 2. F.A.B.
After Removal
of Wax.
No. 3. I.M.
After Removal of
Boric Acid Powder.
No. 4. C.C.B.
After Removal of
Boric Acid Powder.
No. 5. O.O.J.
After Removal of
Boric Acid Powder
No. 6. R.D.
After Removal of
Cotton Plug.
Intensity

Plate 4.

PLATE 4 shows the curves of these cases after the removal of the impacted wax, cotton or borac acid powder. Note that the curves are practically normal.

2. A lowering of the upper tonal limit, especially by B. C. The better the hearing for voice and whisper, the more significant of a lesion of nerve or end organ is the decrease in the upper limit by A. C.

3. Positive Rinne.

PLATE 5 indicates the upper tonal limit in three of the cases of obstruction in the external canal just described by A. C. and B. C. determined with the monochord. Note the upper limit by A. C. is lowered 2,000 d. v. from obstruction in the external canal. There is, however, no cutting down of the upper limit by B. C. and we feel that a lowering of the upper limit by bone conduction is more definite indication of lesion of nerve or end organ than the lowering of the upper limit by A. C.

PLATE 6. The comparison of the upper limit of audition for high notes as determined by A. C. and B. C. is of interest.

The upper chart shows the A. C. and B. C. as determined with the monochord for twenty-two acute suppurative ears. The vertical figures represent the number of cases; the horizantal the upper limit in double vibrations per second. The curves are similar but by air the lowest limit was 12,000 d. v. while by B. C. it was 14,000.

The results of examination of twenty-eight cases of chronic otorrhea are chartered in the center. Again the larger number of cases make similar curves but here, while the lowest limit by B. C. is 13,000 d. v., by A. C. it is 6,000,—a great difference.

Below are graphs secured for charting the results of forty cases of hyperplastic otitis media. Here the similarity of the curves is lost.

The diagnostic significance of the difference between the upper limit as determined by bone and air conduction with the monochord is here illustrated. In cases of acute trouble

Upper limits in cases of occlusion of external auditory meatus.

Before Removal

No. 2. F. A. B. A. C. 17,000 B. C. 19,000

No. 3. C. C. J. A. C. 15,000 B. C. 18,000

No. 4. C. C. B. A. C. 17,000 B. C. 20,000

After Removal

No. 2. F. A. B. A. C. 18,000 B. C. 19,000

No. 3. C. C. J. A. C. 17,000 B. C. 19,000

No 4. C. C. B. A. C. 19,000 B. C. 20,000

PLATE V.

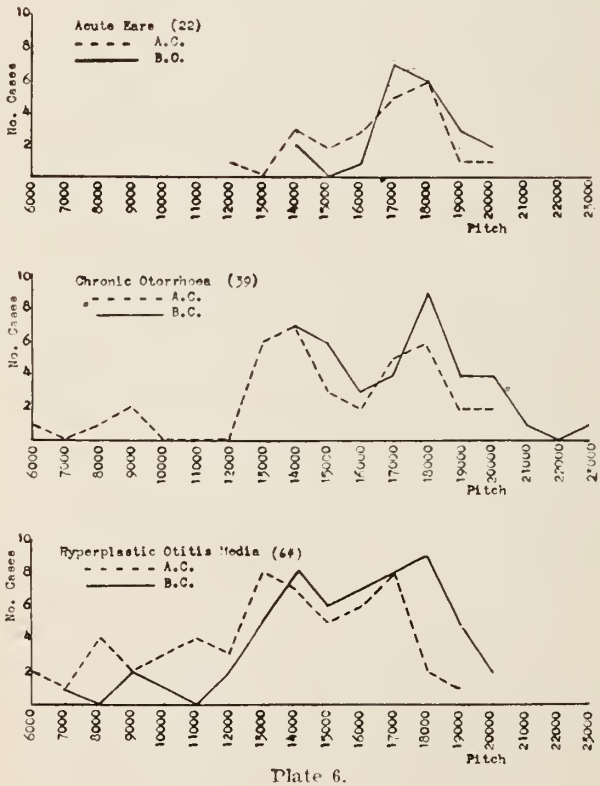

Plate 6.

22% gave a difference of 3,000 d. v. or more between the upper limits determined by air conduction and by bone conduction. In the chronic otorrhea cases this percentage was 39 while cases of hyperplastic otitis media gave this difference in 67%.

The determination of the upper limit by B. C. is a far more accurate indication of the condition of the nerve and end organ than its determination by A. C.

4. The sensitivity for one note or a group of notes is much less acute than the others when determined with the tuning fork or the audiometer.

To determine whether there is a significant difference between the hearing power for high tones and low tones, we have used the audiometer and the Bezold forks. For example, if the hearing for the c^2 fork is indicated by the fraction 20/24 while that for the c^5 fork is indicated by the fraction 5/60, there is a greater decrease for the c^5 than for the c^2. This would be shown on the audiometer record by a curve which closely follows the normal curve for a frequency of 512 d. v. but which falls much below the normal curve at 4096 d. v.

PLATE 7 is a plate of neuro-labyrinthitis showing these distinct depressions in the audiometer curve confirmed by the findings with the tuning forks.

5. Decreased bone conduction as determined by the c^1 and c^2 fork.

This is a very important matter. We feel that a bone conduction decreased two or three seconds is suggestive of a lesion of nerve or end organ when the examination had been properly made. We test bone conduction by placing the tuning fork over the bone just adjacent to the external opening of the bony canal posterior to the upper attachment of the external ear. We keep in mind the influence of mastoid involvement decreasing the bone conduction.

Bezold (Text Book of Otology, page 67) thinks that to be significant the perception by bone conduction for the fork placed on the vortex should be very much shortened.

Wittmaack (Zeit. f. Ohr. vol. 60, page 136) is of the opinion that a shortening of the bone conduction is worthy of diagnosis when it amounts to at least 30% of the average value which we get or receive through the examination of a large number of normally hearing individuals with the chosen tuning fork.

As the percentage of decrease will depend altogether upon the energy with which the fork is excited, this statement is of little value.

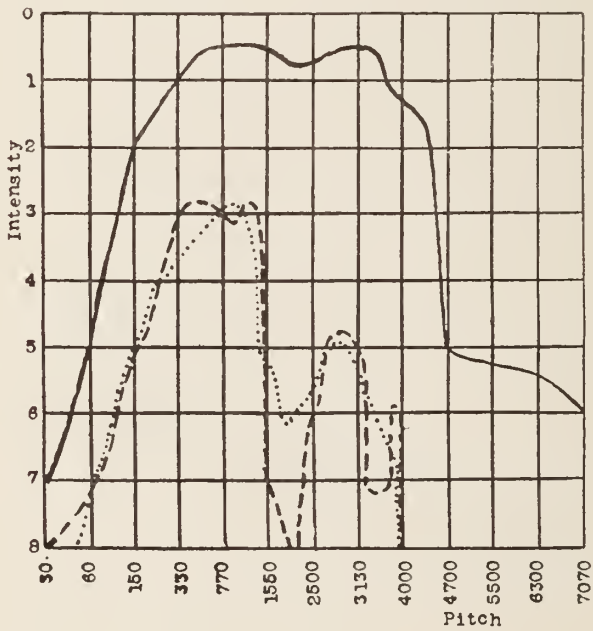

Plate 7.

Bezold feels that an objection to this procedure is that extraneous sounds and the ability of the individual to make an accurate examination and of the patient to cooperate have a decided bearing upon the result. Such criticism is valid for all functional tests of hearing and is as true for determining hearing power by A. C. as well as B. C.

Our examinations are conducted in a room set apart especially for this purpose and, while it cannot be said to

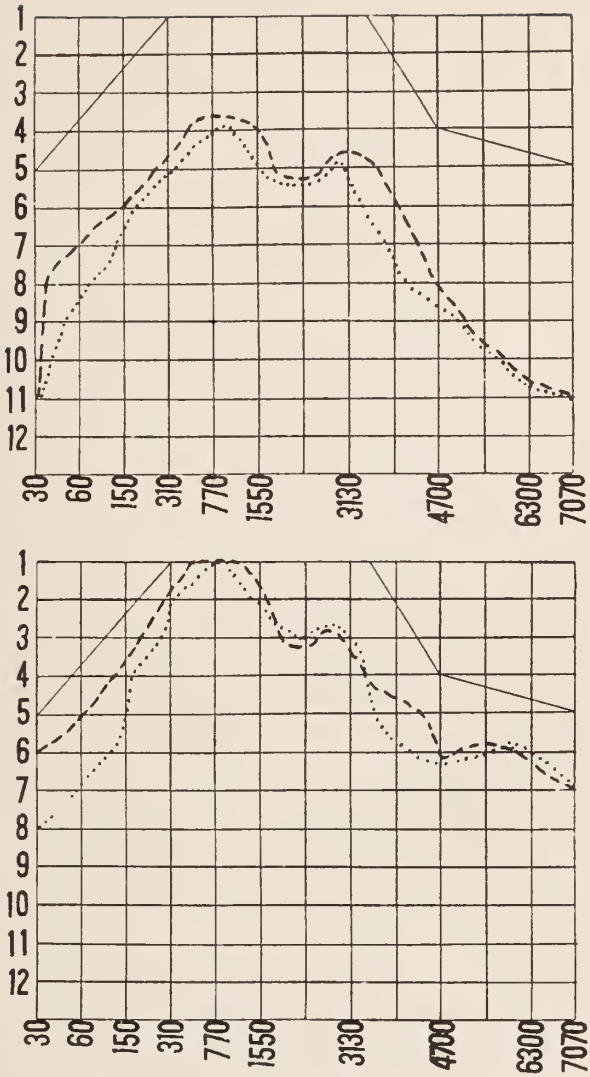
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Plate 8.

be entirely free from external noises, it is probably much more nearly ideal for this purpose than those ordinarily used.

Emerson (Annals of O. R. & L. Dec. 21, page 999), is of the opinion that when B. C. is lowered for the 256 or 512 d. v. fork that nerve deafness has been present for a long time.

Werhofsky (Prufung, d. Hordauer in Verlaufe der Tonskala bei Erk. d. Mit. u. Innern O. Bezold U. d. Funk. Pruf. d. Mench. Gehor, 1897-1909) found decreased B. C. in all lesions of the nerve or end organ that he studied. He reports B. C. decreased two seconds in one case of nerve deafness and three seconds in another.

He says that B. C. is less with higher forks than with lower and cites a number of observations to confirm this.

In describing his technique he does not state the length of time his forks vibrate with a given stimulus. Our higher forks vibrate longer than the lower and consequently the decrease in B. C. with the higher forks would be greater.

Siebenmann in an article translated by Dench (Archives of Otology, vol. 22, Pg. 5) says that in apparently normal ears there may be functional defects in the test for the upper tone limit and in the perception of sound by B. C. He also shows that B. C. is sometimes decreased by aspiration of the middle ear.

Page, (Proceedings N. Y. Academy of Med. Oct. 13, 1921) says that the early cases of otitis media have a negative pressure in the tympanum.

Kerrison (Text Book of Otology, Pg. 89) suggests that the hearing by bone should be decidedly decreased to be significant.

Schwabach (Uber Harprufung, etc. Archiv. f. Ohren. vol. 31, page 6) says that B. C. is longer than with normal persons in lesions of the transmitting apparatus while it is the same as normal persons or less in lesions of the perceiving apparatus of the ear.

In our study of acute middle ear infections, we have found it very unusual to find an infected ear without evidence of a lesion of its nerve or end organ.

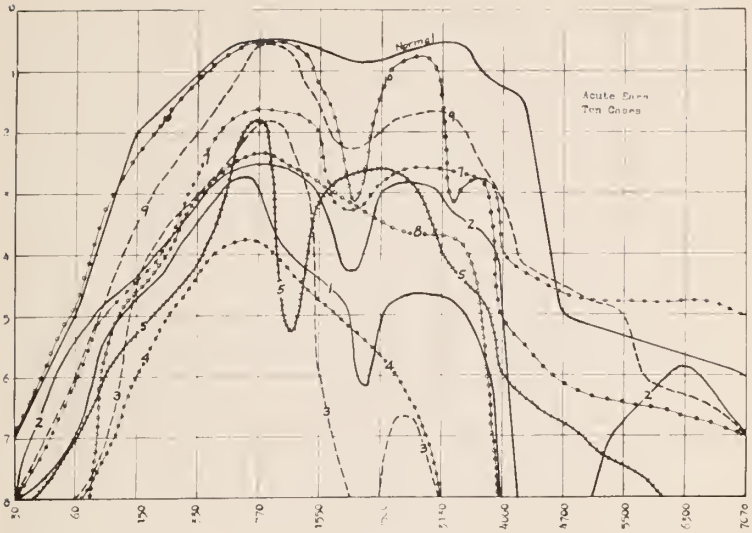


Plate 9.

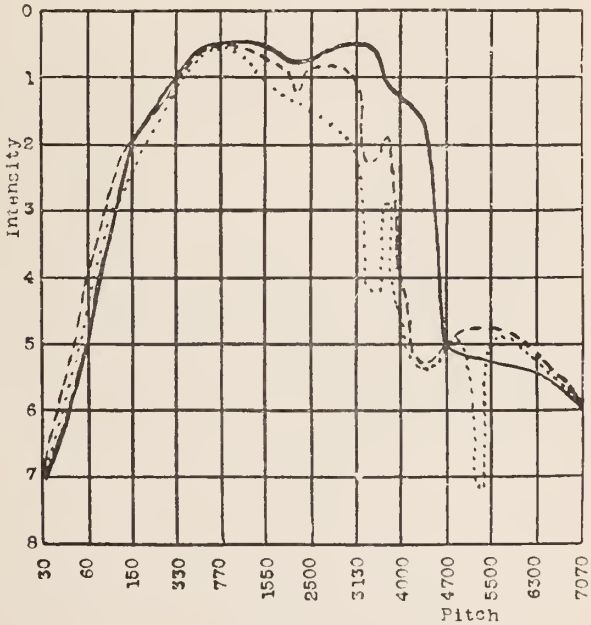


Plate 10.

Siebenmann, in an article translated by Dench (Archives of Otology, Vol. 22, Page 12) reports that in making functional examinations of pure cases of catarrh of the eustachian tubes, he found the upper tone limit usually lowered and not restored by inflation. He assumed that the lowering of the upper limit was due to a passive hyperemia of that portion of the scala vestibuli lying next the oval window.

PLATE 8 shows curves of a case of acute tubal catarrh where we found no definite evidence of lesion of nerve or end organ. The decrease in hearing for the voice and whisper was apparently due wholly to the middle ear condition and disappeared on inflation. B. C. in each ear was normal. Upper limit by Monochord, R., A. C. 16,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000. As with the blocking of the external canal the upper limit was lowered by air and not by B. C. Unfortunately, after inflation the upper limit was not tested by A. C.

The curve before inflation shows rather a uniform decrease in audition for all notes except the very lowest which were not heard at all. The audition for the notes became almost normal on inflation.

Twenty-two acute infected ears were studied. Of the twenty-two, fourteen had positive Rinne and eight had negative Rinne; fifteen had decreased bone conduction for the c^2 fork and fourteen had the upper tone limit decreased 2,000 or more by A. C. as determined by the monochord. Thirteen of the fourteen had the upper limit by B. C. decreased 2,000 or more. Twelve cases had decreased bone conduction as well as lowered upper limit. Two cases had decreased B. C. with upper limit normal and two cases had upper limit by A. C. lowered but B. C. normal.

PLATE 9 shows the curves of ten acute infected ears. The dots, dashes and circles in the curves are added so that they may be more easily followed through the entire field. You will note that curves No. 2 and 3 show complete gaps. Partial gaps are shown in all the curves except No. 4. 90%

of the curves of acute infected ears are suggestive in this manner of lesions of the nerve or end organ.

A careful analysis of the clinical charts of the twenty-two ears studied shows that in every acute infected ear except one there was either decreased B. C., upper limit lowered by B. C. 2,000 or more, or a defect in the audiometer curve suggestive of lesion of nerve or end organ.

These evidences of lesion of nerve or end organ appear very early in the course of the disease.

PLATE 10. H. B. Examination was made within twelve hours after first evidence of infection. With good hearing

Unaffected Ears in Unilateral Cases.

Name	Rinne	B. C. (c_2 fork)	Monochord A. C.	Monochord B. C.
A. A. S.	+	Normal	19,000	19,000
M. K.	+	Normal	15,000	17,000
G. V. B.	+	Normal	16,000	17,000
E. C.	+	—	17,000	18,000
B. D.	+	—	18,000	18,000
E. S.	+	—	19,000	21,000

PLATE 11.

for voice, spoken, 45 feet, and whisper, 15 feet, in each ear; frank earache in the left, slight earache in the right; drum-heads reddened, no bulging; Rinne positive in each ear, c^5 fork in left 30/60; with other forks, normal. Two partial tone gaps in left ear with audiometer curve. B. C. -2 sec. each; upper limit lowered 2,000 by B. C. with monochord. No leucocytosis at this time. Patient discharged ten days later, B. C. normal. c^5 heard normally,—upper limit normal by A. C. and B. C.

In cases where the infection was unilateral the evidences of a lesion of nerve or end organ were not confined to the infected ear.

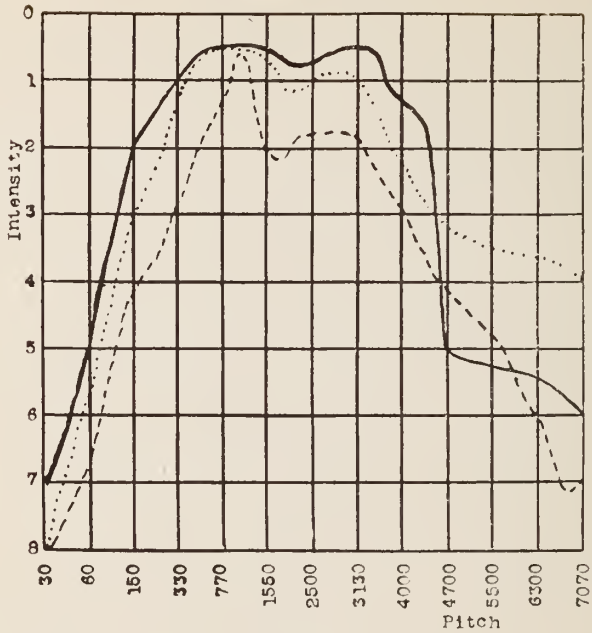


Plate 12.

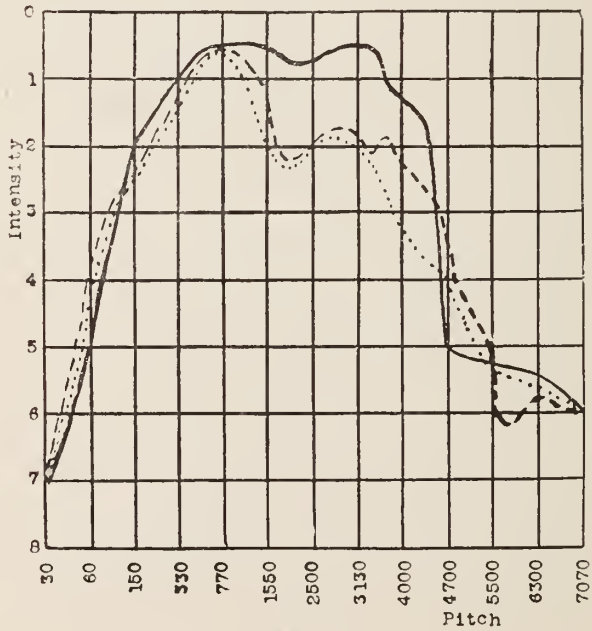


Plate 13.

PLATE 11 shows the findings of the good ear in six cases of unilateral acute infection of the middle ear. Only one case, the first, had no suggestion lesion of end organ or nerve in the good ear. In the second, third, fourth and fifth cases the upper limit is lowered; in the fourth, fifth and sixth B. C. is decreased. The audiometer curves of the first case, and of cases three and four, were somewhat suggestive of lesion of nerve or end organ so that in at least five out of six of the unaffected ears and probably all six, there is evidence of involvement of the nerve or end organ.

PLATE 12 B. D. This case is interesting because a few days before infection of the left ear the ears were tested and were apparently normal. Note that in the right ear where there was no redness of drumhead or other evidence of middle ear infection, the decreased hearing for voice and whisper, the decreased B. C., the decreased upper limit by B. C., and the slight diminution of audition for the c^1, c^2, c^3, c^4, and c^5 forks, for the left ear, the decreased B. C., the tuning fork tests and the audiometer curve are all suggestive of lesion of nerve or end organ.

Treatment and eradication of the middle ear condition may have no influence upon the lesion of nerve or end organ.

PLATE 13 E. B. shows the findings at the heighth of an acute tubal catarrh. The tubal orifices were edematous covered with dilated blood vessels; the drumheads were reddened, not bulging. B. C. decreased each 2 sec. Upper limit each B. C. 17,000 by monochord.

PLATE 14 shows the findings after the eradication of the middle ear lesion. B. C. -3. Upper limit lowered by B. C. Audiometer curve indicative of lesion of nerve or end organ. The findings remained the same as long as the patient remained under observation.

PLATE 15 shows the findings A. A. S. Feb. 27, taken one month after an infection of the left ear was apparently well. Curve and tuning fork findings for left ear indicate

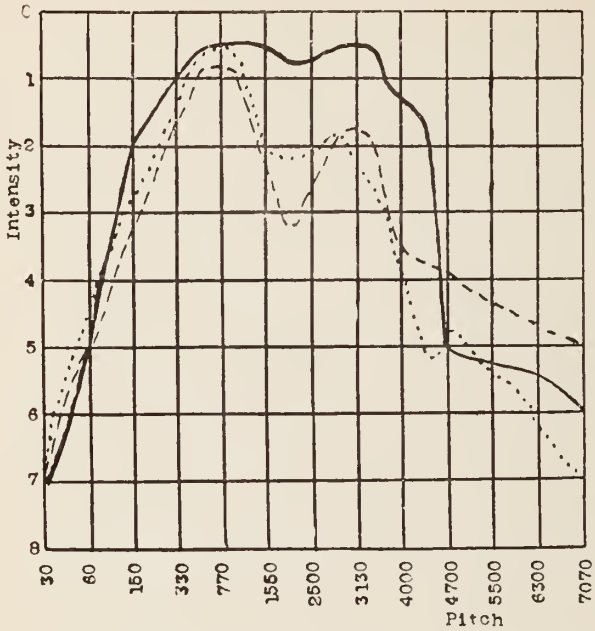


Plate 14.

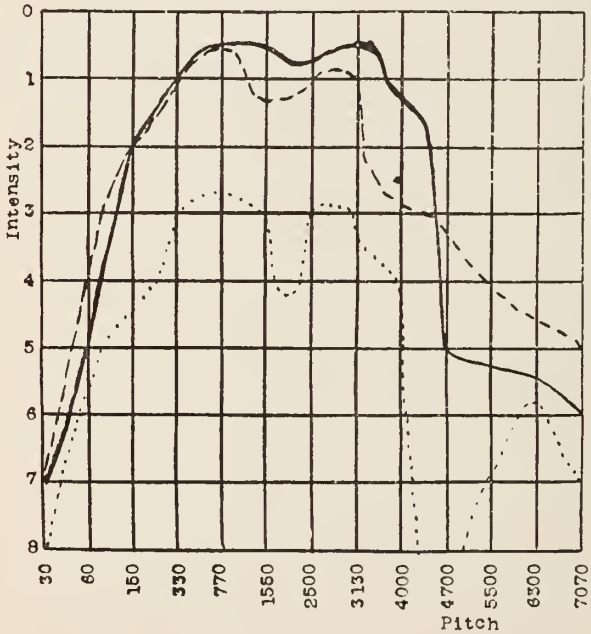


Plate 15.

a lesion of importance involving nerve and end organ. Whisper and voice are poorly heard.

PLATE 16 B. D. Sept 27, shows findings in both ears in a case of acute infection of the left ear one month after discharged as cured. The curve left, tuning fork tests left, and decreased B. C. each indicates a lesion of nerve or end organ left and probably also right. At no time during the disease was there evidence of acute middle ear infection right. Nevertheless, the hearing right which was W15, V45, before the infection of the left ear is now W13, S45, while the B. C. is -5. Before the infection of the left ear it was normal.

After acute infections of the middle ear with return to normal audition for voice and whisper there may be definite indications of lesions of nerve or end organ.

Twelve cases of bilateral chronic otorrhea and fifteen cases of unilateral chronic otorrhea, or thirty-nine ears, suffering with chronic otorrhea, were studied. Rinne was negative in thirty-five ears, positive in four. There was decreased bone conduction in eleven ears, increased in twenty-six, normal in two. Decreased B. C. was not so common as in the acute infections. The upper tone limit was decreased 2,000 or more by bone conduction in thirty ears. In thirty-seven ears the audiometer curve was suggestive of a lesion of nerve or end organ and in two ears not. Marked depressions and tone gaps in the audiometer curves were much more frequent than in the acute ears. The curves were much more characteristic of lesions of the nerve or end organ. If we include the results with the tuning fork tests in only one ear out of the thirty-nine, was there no evidence of a lesion of the nerve or end organ.

PLATE 17 R. B. is a typical chart showing the findings in a case of chronic otorrhea. Note that with the tuning forks the hearing range is from c^1 to c^4, inclusive. The audiometer curve shows the decreased audition for both low and high notes in the range of the audiometer with depressions characteristic of nerve or end organ. The upper limit in each ear is decreased to 15,000 d. v. by B. C.

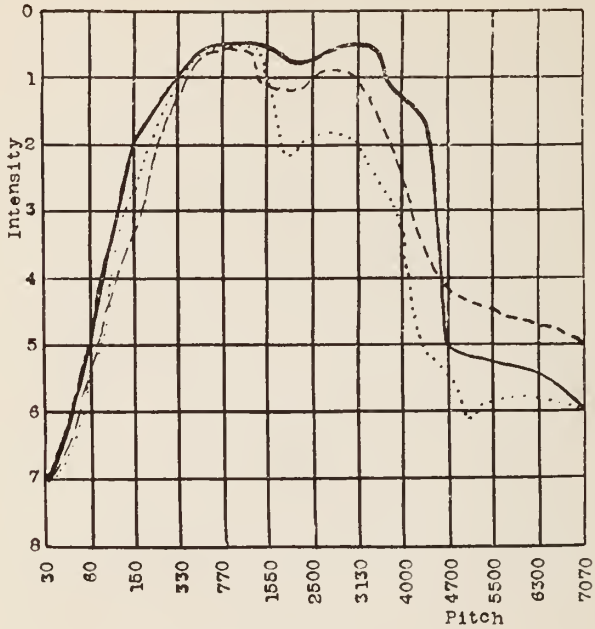


Plate 16.

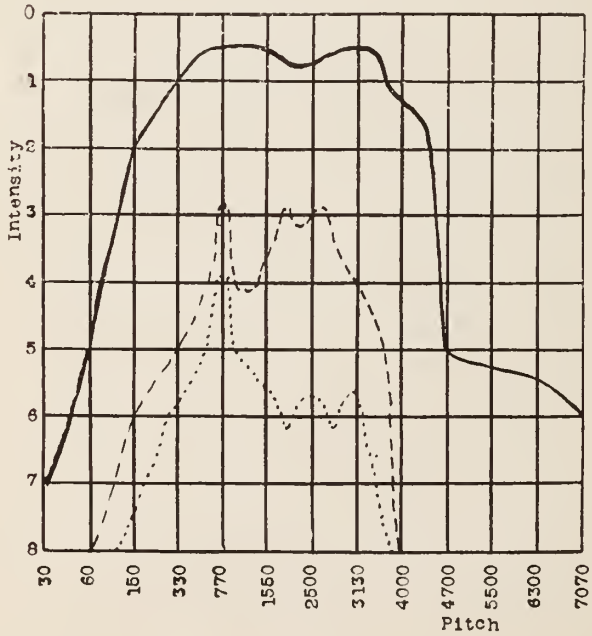
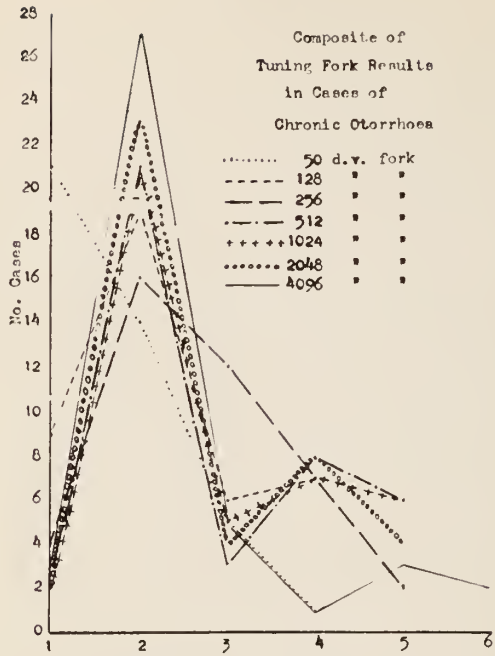


Plate 17.

From a study of the cases of chronic otorrhea one gets the impression that the extent of the lesion of nerve or end organ corresponded roughly with that of the middle ear lesion, while in the acute cases there was the greatest variation between the extent of the lesions of the inner ear and those of the middle ear. It was not unusual with a very mild acute middle ear infection to have a very well marked lesion of the nerve or end organ. A study of the fifteen unaffected ears in the fifteen cases of unilateral chronic otorrhea shows that Rinne was negative in unaffected ears no times, positive in fifteen. B. C. was decreased in the unaffected ears three times; it was normal ten times and increased twice; the upper limit was decreased by B. C. 2,000 or more five times and was normal ten times. The audiometer curve of the unaffected ears was indicative of a lesion of nerve or end organ in only one case. The unaffected ears in this series of unilateral cases were very good ears with one exception. The hearing in almost every ear was W15 feet. While these findings suggest that there are a fair number of lesions of the nerve or end organ in the unaffected ears in chronic otorrhea we do not find the large percentage of involvement of the nerve or end organ as in the unaffected ears in the acute infections. This suggests to us that the involvement of the nerve or end organ in the unaffected ears in acute infections is frequently not a permanent affair; otherwise, we would find the same number of lesions in unaffected ears in chronic otorrhea. The lesions of the nerve or end organ in ears suffering from chronic otorrhea were much more marked than in the acute infections. There is a direct interdependence between the chronic otorrhea and the lesions of the nerve or end organ. The chronic otorrhea has no influence of any moment upon the nerve or end organ of the opposite side. In our acute cases we get the impression that the acute middle ear trouble and the lesion of the nerve and end organ are not interdependent. They may both be due to the same cause but not necessarily so.



1 Not Heard
2 Heard by a fraction equivalent to or less than 1/4
3 " " " " between 1/4 and 1/2
4 " " " " " 1/2 " 3/4
5 " " " " " 3/4 " 1/1
6 Normal

Plate 18.

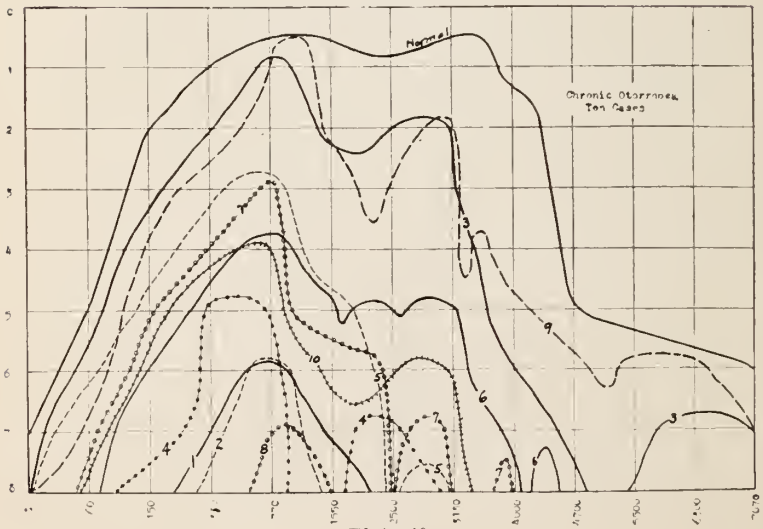


Plate 19.

PLATE 18 is a composite graph of the results obtained from tuning fork examinations. It is difficult to express in a uniform manner the results obtained in tuning fork examinations when different denominators are used to represent normal hearing for different tuning forks. For example; in measuring the hearing power for the tuning forks we represent the hearing for a normal ear in terms of distance as the denominator of a fraction. The hearing power for that fork by a pathological ear is recorded in the same terms in the numerator. For the c^5 fork, since a very slight impact produces a sound heard at a considerable distance, we use the denominator 60. The 256 fork is heard at a less distance and we use 24 for the denominator. For similar reasons we use the denominator 4 with the 50 d. v. fork.

In order to make an analysis of our results we have arbitrarily divided our results into six classes; those who do not hear the fork at all, are put in class 1, those who hear the fork when it is very loud as indicated by fractions equivalent to one-fourth or less we have put in Class 2, those who hear it by values indicated by the fractions between one-fourth and one-half are put in Class 3; those who hear it by fractions from one-half to three-fourths are put in Class 4; those who hear it from three-fourths to less than normal are put in Class 5; and those who hear it normally are put in Class 6. The curve shows that twenty-one cases did not hear the 50 d. v. fork at all; three did not hear the c^5 fork; fourteen heard the 50 d. v. fork with a fraction equivalent to one-fourth or less; while twenty-seven heard the c^5 fork with results indicated by a fraction of one-fourth or less. You will notice that the 512 d. v., 1024 d. v. and 2048 d. v. forks run almost parallel throughout the entire curve while the greatest variations in chronic otorrhea cases are shown with the 50 d. v. fork and the 4096 d. v. fork. There were no cases where the hearing was perfect for the low fork and two cases where the hearing was normal for the 4,096 d. v. fork.

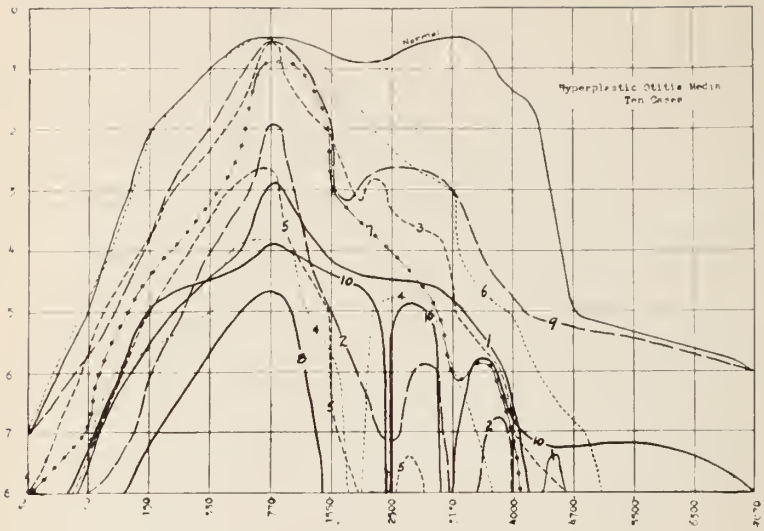
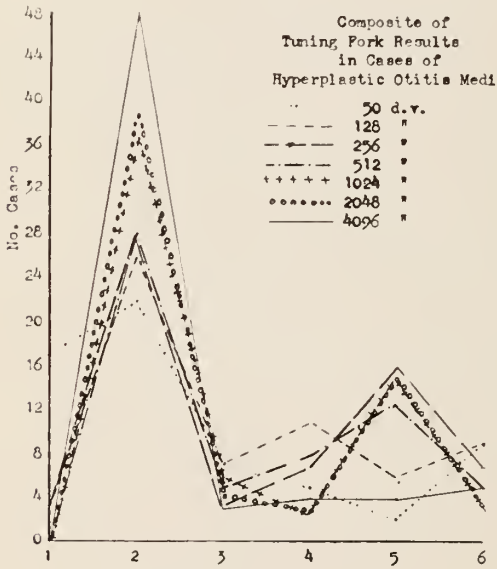


Plate 20.



1 Not heard
2 Heard by a fraction equivalent to or less than 1/4
3 " " " " between 1/4 and 1/2
4 " " " " " 1/2 " 3/4
5 " " " " " 3/4 " 1/1
6 Normal

Plate 21.

PLATE 19 shows the results of the audiometer examinations in ten cases of chronic otorrhea. It is inserted to show the frequency with which such phenomena as tone islands, gaps and other peculiarities exist. These peculiarities are indicative, in our judgement, of lesions of the nerve or end organ. For example, curve No. 8 shows simply a region of residual hearing for tones between 500 d. v. and 1,500 d. v. No. 7 while rising much higher on the chart, shows two islands with the interlying gaps at 2,500 d. v. and at about 3,200 d. v., Curve No. 9, even though rising to the normal at 1,000 d. v., shows that the low tones are lost, that there is a decrease for notes above 1,200 d. v. and a return to almost normal at 5,500 d. v.

Sixty-six percent. of the records of cases with chronic otorrhea show either complete or marked partial tone gaps. These do not include such cases as are illustrated by curves No.1, 2, and 8 which are equally significant of an inner ear lesion.

In only one case L. P. age 13 did a tone gap disappear with treatment.

Hyperplastic Otitis Media. Thirty cases of bilateral H. O. M. and four cases of unilateral H. O. M., or sixty-four ears were studied. Rinne was negative in thirty-three ears, positive in thirty ears, questionable in one ear. There was decreased bone conduction in forty-four ears, increased bone conduction in sixteen ears, normal in four ears. The upper limit was decreased by B. C. 2,000 or more d. v. in forty-nine ears.

Twenty-one ears had complete tone gaps in the audiometer findings. In eight more there were marked partial gaps or 45% showing these peculiarities. In addition to this there were 34 cases where there were marked depressions equally indicative of inner ear involvement.

PLATE 20 shows the curves of ten typical cases of H. O. M. Complete tone gaps are illustrated in curve No. 10 which has a gap at 2,400 d. v. and another at 2,000. Curve No. 9 shows a dip or partial gap at 1,600 d. v. No. 6, while not showing either a partial or complete gap shows that the

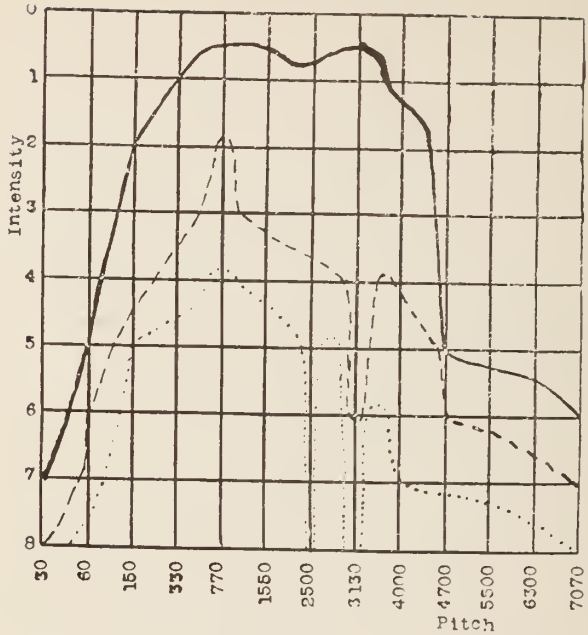


Plate 22.

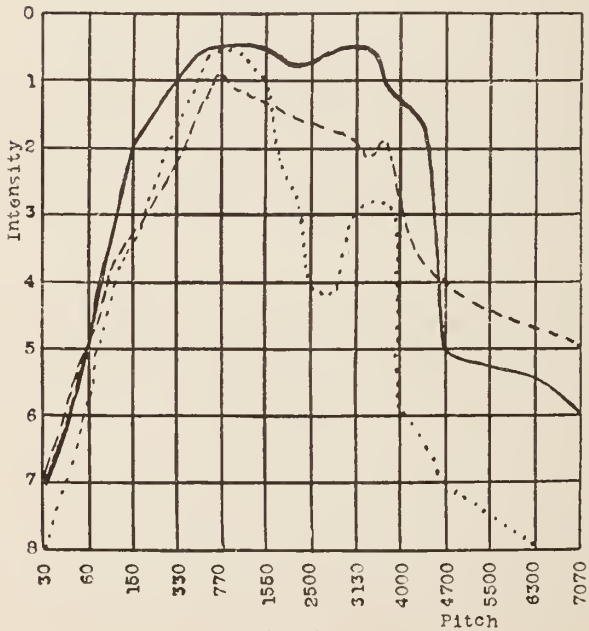


Plate 23.

hearing was normal up to 1,500 d. v. and decreased more and more for tones higher in the scale. A curve of this type certainly indicates a lesion of the nerve or end organ. Curve No. 8 shows only a patch of residual hearing between 100 d. v. and 1,500 d. v. Each curve shows marked diminution in the audition for the c^5 fork. In the next plate you will note that in only eight of the sixty-four ears was the c^5 fork heard normally.

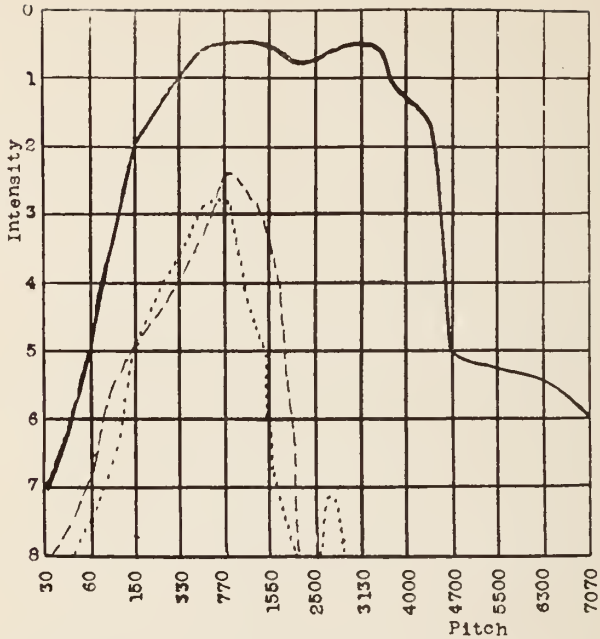
PLATE 21 shows a composite of the tuning fork cases in hyperplastic otitis media. Here we have made the same arbitrary divisions as we did with Plate No. 18. Here again we see that the larger variation is shown with the 4096 d. v. and the 50 d. v. forks. The 1024 d. v. and the 2048 d. v. forks run almost parallel through the entire range. While there were no cases where the 4096 d. v. fork was not heard there were 51 cases who could hear this fork only when it was struck very loud as, for instance, when the fork was forcibly struck against the heel of the shoe, twenty cases did not hear the 50 d. v. fork.

Note that in eight cases the 50 d. v. fork was heard apparently normal. In these eight cases the lesion of the nerve or end organ over-shadowed the middle ear lesion.

A study of the clinical charts of these cases shows evidence of the involvement of the nerve or end organ in every ear affected save one.

PLATE 22 F. K. shows the curves of a bilateral case of H. O. M. Note the negative Rinne, slightly decreased B. C., power of hearing low and high notes markedly interfered with in each ear, tone gaps in the audiometer curve, and upper limit markedly decreased both by A. C. and B. C.

PLATE 23 C. L. shows in the right ear bone conduction normal, and negative Rinne, audiometer curve practically normal, upper limit normal. In the left ear B. C. is decreased five seconds. The right ear of this case was the only one of the sixty-four ears studied that did not show some evidence of involvement of nerve or end organ. Hearing was poorer in the right ear.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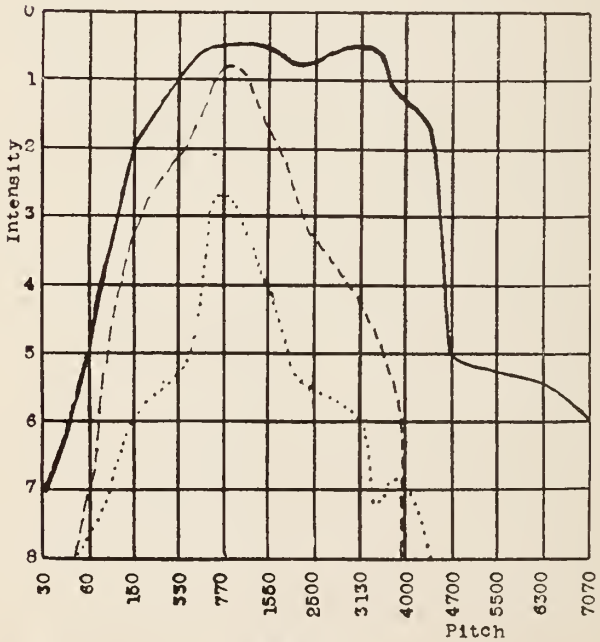
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Plate 24.

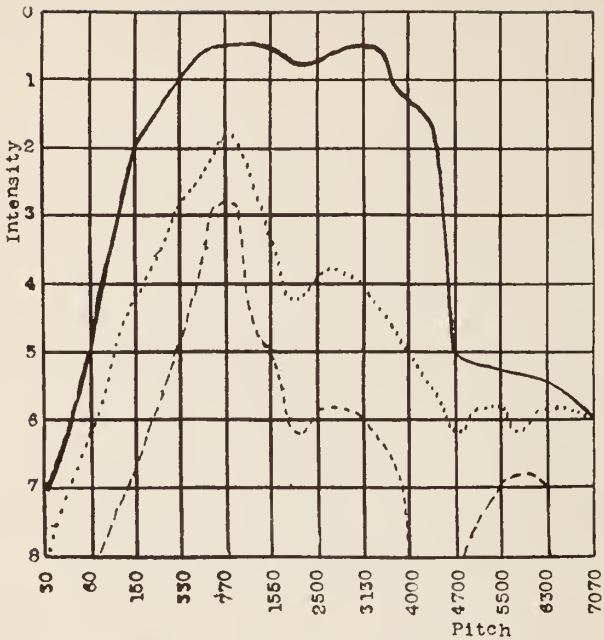
There was found no definite relation between the extent of the inner ear lesion as compared with the middle ear lesion.

PLATE 24 shows the findings in H. S. aged 62, and in C. P. aged 62. The objective findings of the middle ear were similar. In one case the bone conduction is -5 seconds and in the second case it is -12 seconds. In the first case, the audiometer curve shows a tone gap in the left ear and in the second case there is none. In the audiometer curve the upper notes are heard much poorer in the first than in the second. In the first case the upper limit is so low that it cannot be detected using the Galton whistles or the monochord either by A. C. or B. C. and with the Koenig cylinders it is only approximately half normal 21,000 s. v. instead of 41,000, while in the second case the upper limit is decreased, it is 16,000 by B. C. with the monochord. The lesion of the nerve or end organ is much more marked in the first than the second case. In the second case, the one with the least involvement of the nerve, had a positive Wassermann, the first one not.

PLATE 25 (Findings of Miss D. and H. B.)

The age of the first is twenty-five years and the age of the second is twenty years. The hearing in the left ear is approximately the same for whisper. In the first case B. C. left is increased, the tuning fork test shows fair hearing for the c^4 and c^5 fork, the upper limit by B. C. is normal, the audiometer curves, while there is a gap in the right ear and depressions in the left shows fair hearing for some of the notes in the higher part of the range. In the second case, however, B. C. for c^1 fork is -5, c^2 fork -6, hearing for the c^4 and c^5 fork is markedly decreased, the audiometer curves show a cutting down of the higher notes to 4,000, the upper limit by B. C. is 15,000. The evidence points to a much more marked lesion of the nerve or end organ in the second case than in the first.

In one case of H. O. M. while under observation acute suppurative otitis developed. With the diminution in hearing the B. C. which was plus 4 in the left ear became de-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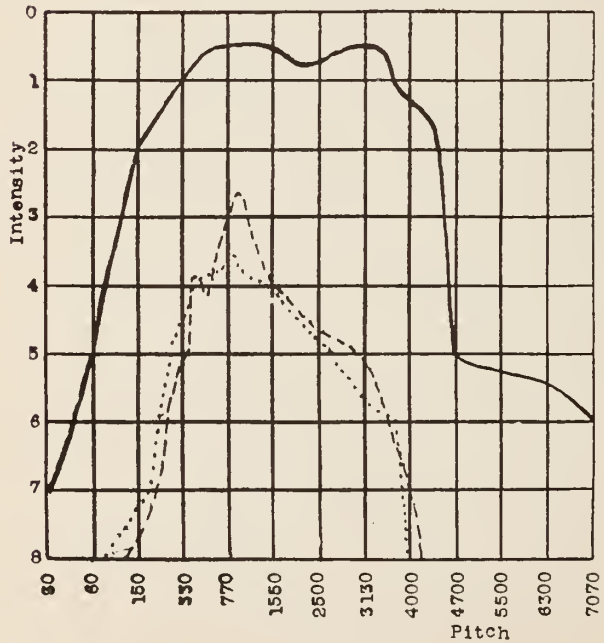
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Plate 25.

creased. With the disappearance of the acute otitis it became plus again.

Our cases of H. O. M. frequently tell of improvement in hearing that is satisfactory to them following one kind of treatment or another. Our tests have failed to show any actual improvement in any case unless the lesion of the nerve or end organ greatly over-shadowed the middle ear lesion and the improvement resulted from the treatment of the case for syphilis, or removal of tonsils, or whatnot.

PLATE 26. H. J. is a case of H. O. M. where there was also a plus 4 Wassermann. Note the positive Rinne with the c^2 fork and the greatly decreased B. C. with the same fork and a negative Rinne with the c^1 fork which is decreased only six seconds. This patient's deafness has rapidly appeared in the last six months. It has progressed too rapidly for H. O. M. The syphilitic lesion of the nerve or end organ is more responsible for the deafness.

PLATE 27. J. C. is a case of H. O. M. with negative Wassermann and no evidence of syphilis where the lesion of nerve or end organ greatly over-shadowed the middle ear lesion. Note the Rinne plus, B. C. decreased 6 seconds and 4 seconds, the tuning forks suggestive of lesion of nerve or end organ. The upper limits by B. C. 12,000 d. v. and 13,000 d. v. The curve is also characteristic of lesion of nerve or end organ.

A study of the unilateral cases of H. O. M. gives some very interesting information. Four cases were studied. In the unaffected ear. negative Rinne in no cases. Decreased B. C. in two cases, upper limit decreased 2,000 or more by B. C. in three cases. In all four of the unaffected ears there was some evidence of lesion of nerve or end organ if we include the findings with tuning forks and audiometer.

Conclusions:

I. The determination of the upper limit by B. C. is a far more accurate indication of the condition of the acoustic nerve and its end organ than its determination by A. C.

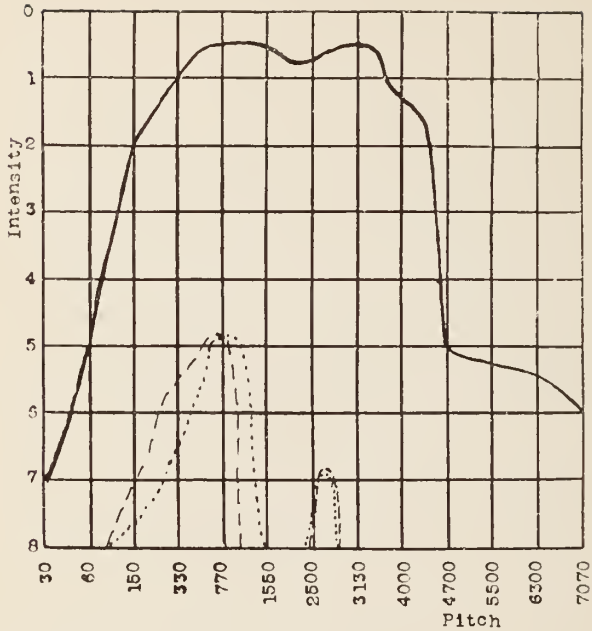

Plate 26.

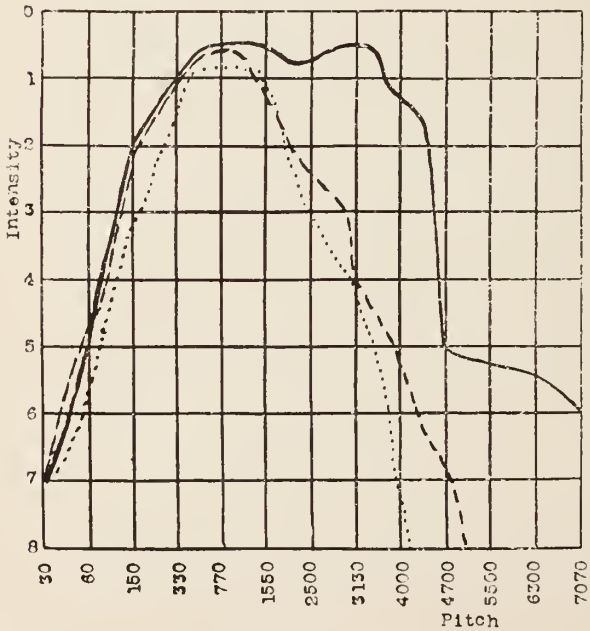

Plate 27.

II. It is usual in acute middle ear infections even in the early stages to find evidences of involvement of nerve or end organ. Rarely with the present methods of diagnosis are we unable to find evidence of such involvement.

III. The lesion of nerve or end organ is not necessarily dependent upon the acute middle ear infection. There is no definite relation between the severity of the middle ear infection and the lesion of nerve or end organ. When the middle ear infection is unilateral the unaffected ear usually presents evidence of a lesion of nerve or end organ.

IV. After the disappearance of the acute middle ear infection there may remain a permanent lesion of nerve or end organ.

V. In every case of chronic otorrhea except one there was evidence of a lesion of nerve or end organ. There is an interdependence between the middle ear lesion and that of the nerve or end organ.

VI. In the H. O. M. cases every ear studied save one presented evidence of lesion of nerve or end organ.

VII. In H. O. M. there is no constant relation between the extent of the lesion in the middle ear and the lesion of nerve or end organ.